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## Effects of noise overexposure on distortion product otoacoustic emissions

*Auditory hazard from impulsive occupational exposures*

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THESIS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

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Effects of noise overexposure on distortion  
product otoacoustic emissions

– Auditory hazard from impulsive occupational exposures –

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Aalborg, Denmark 2010



# Preface

This thesis is submitted to the Faculty of Engineering and Science at Aalborg University in partial fulfillment of the requirements for the Ph.D. degree. The work has been carried out in the time period October 2005–2008 at the Section of Acoustics, Department of Electronic Systems, Aalborg University.

The thesis is organized as an accumulation of papers consisting of two journal papers and one conference paper. The thesis starts with a general description explaining the scope of the investigation, the background theory supporting this work and the experimental approach. The main results are also presented and discussed. The papers are included at the end of the thesis.

Financial support by the STVF framework programme 97011320 *Research in human sound perception - with special reference to electroacoustic applications* - sponsored by the Danish National Research Council, is sincerely acknowledged.

I would like to express my most sincere gratitude to the following people who supported me during my PhD work: First of all, to my supervisor professor Dorte Hammershøi for her extraordinary ability to challenge myself both professionally and personally. For her constant guidance, scientific and technical expertise, discussions and sincere criticism. Secondly, to my co-supervisor associate professor Rodrigo Ordoñez who has supervised my work since my early days as a master student at Aalborg University. From Rodrigo I admire his scientific curiosity, his passion and academic enthusiasm. I always enjoyed the discussions with Rodrigo because he always came up with new perspectives that sharpened up my own knowledge. Special thanks to associate professor Karen Reuter. This work would not have been possible without her previous and invaluable work on otoacoustic emissions. I also thank Karen for her remarkable experience and helpful advices.

Special thanks to all the staff at the Section of Acoustics at Aalborg University, also those who left. I was a foreigner studying abroad but I always felt at home.

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Finally, I would like to thank all the subjects who participated in the experiments for their constant positive attitude

Miguel Angel Aranda de Toro

Copenhagen, 2010



# List of papers

- **PAPER I:** M. A. Aranda de Toro, R. Ordoñez, K. Reuter and D. Hammershøi, *Recovery of distortion product otoacoustic emissions after a 2-kHz monaural sound-exposure in humans: effects on fine structures*. Accepted for publication at the *Journal of the Acoustical Society of America* with manuscript number MS# 10-08995R (accepted 30th of September, 2010).
- **PAPER II:** M. A. Aranda de Toro, R. Ordoñez, K. Reuter and D. Hammershøi, *Is it necessary to penalize impulsive noise +5 dB due to higher risk of hearing damage?*. Submitted for publication to the *Journal of the Acoustical Society of America* and currently in the second revision process with manuscript number MS# 10-08966R (last submitted 24th of September 2010).
- **PAPER III:** M. A. Aranda de Toro, R. Ordoñez and D. Hammershøi, *Optimization of distortion product otoacoustic emission (DPOAE) measurements with the system ILO96*, Proceedings of 19th International Congress on Acoustics -ICA07-, Madrid: Spanish Acoustical Society, volume ID0943, 92–104 (2007).



# Summary

Industrial workers are often exposed to noise levels that can damage their hearing. The risk of noise-induced hearing loss (NIHL) can be predicted according to the International Standard ISO 1999:1990. Unfortunately, the validity of the method established by the standard to correctly predict the risk of hearing loss for all types of noise exposures is still under question.

One controversial aspect refers to noises of impulsive character. The current conception is that the risk of NIHL from all types of existing noise in industrial environments can be predicted on an energy basis. For this reason, the standard establishes the same assessment method for all types of noise, also impulsive, based on readings of the total A-weighted acoustic energy of the noise within the duration of the exposure ( $L_{Aeq,T}$ ), normalized to a normal working day of eight hours  $L_{EX,8h}$ . Several studies have shown that for equal amounts of energy impulsive noise may be more dangerous for our hearing than continuous noise. For this reason, ISO 1999:1990 allows adding a +5 dB penalty when measuring noises of impulsive character based on the presumption that they might pose a higher hazard of hearing loss. Nevertheless, the penalty is a precautionary measure and currently there is not enough systematic data to demonstrate its validity –nor to determine whether all types of impulse noise must be penalized.

The evaluation of hearing ability and diagnosis of hearing loss is typically performed with standardized pure-tone audiometries. However, otoacoustic emissions (OAEs) may provide a more sensitive diagnosis tool to evaluate risk of NIHL. Longitudinal and cross-sectional studies have shown that OAEs can diminish before hearing levels in populations exposed to high levels of noise – which suggests that changes in the properties of OAEs could be preclinical indicators of NIHL. Unfortunately, our understanding of OAEs is not complete and researchers are still investigating how to optimize OAE measurements in order to provide the maximum information regarding the healthy functioning of the inner ear and its vulnerability to noise.

In the present dissertation, 16 normal-hearing human subjects were exposed to moderate noise-stimuli under laboratory conditions in two different experiments. The effect of the noise stimuli on the hearing of the subjects was monitored with measurements of distortion-product otoacoustic emissions (DPOAEs). The experiments were restricted to evaluate temporary changes on the characteristics of the DPOAEs - and therefore reversible. The underlying mechanisms between temporary and permanent auditory changes are likely to be physiologically different. For this reason, our results cannot be directly extrapolated to prolonged exposures in real industrial settings. However, the presence of a higher temporary change in one of the stimuli may be indicative of a higher risk of NIHL.

The experiments had the following purposes:

1. To investigate the validity of the +5 dB for impulsiveness for predicting risk



of NIHL. Subjects were exposed for 10 minutes to two types of binaural industrial-recordings: (1) a continuous broad-band noise normalized to  $L_{EX,8h} = 80$  dBA; and (2) the combination of the previous stimulus with an impulsive noise normalized to  $L_{EX,8h} = 75 + 5_{dB_{penalty}} = 80$  dBA (peak level 117 dBC and repetition rate of 0.5 impacts per second). DPOAEs were measured in a broad frequency range before and in the following 90 minutes after the exposure. The assumption is that, if the penalty is correct, both stimuli may produce a similar effect on the DPOAEs.

2. To investigate methodological aspects of DPOAE measurements that might improve hearing diagnosis and detection of hearing loss. In particular, it was studied whether the fine structures of the DPOAE are systematically affected after a tonal overexposure; and whether the evaluation of the fine structures might provide more information about the vulnerability of the inner ear than the DPOAE level alone. Subjects were exposed monaurally during 10 minutes to a 2 kHz tone normalized to an exposure level  $L_{EX,8h}$  of 80 dBA. DPOAEs were measured before and in the following 70 minutes after the exposure. The experimental protocol allowed measurements with high time and frequency resolution in a 1/3 octave-band centered at 3 kHz.

The results indicate that:

1. The continuous exposure had a bigger impact on DPOAE levels, with a maximum DPOAE shift of approximately 5 dB in the frequency range 2 – 3.15 kHz during the first 10 minutes of the recovery. No evident DPOAE shift is seen for the impulsive+continuous exposure. The results indicate that the penalty overestimated the effects on DPOAE levels and support the conception that the risk of hearing loss from low-level impulses may be predicted on an equal-energy basis.
2. The fine structures of the DPOAE are highly individual and no systematic change was observed after noise overexposure. Therefore, the evaluation of the fine structures might not be a better indicator of risk of hearing loss. On the contrary, the differences across subjects can complicate the interpretation and comparison of results.

# Resumé

## **Virkningerne ved overeksponering for støj på *Distortion Product Otoacoustic Emissions* – Risikoen for høreskader ved eksponeringer for impulsstøj på arbejdspladsen**

Industriarbejdere er ofte udsat for støjniveauer, der kan beskadige deres hørelse. Risikoen for støj induceret høretab (*noise-induced hearing loss*, NIHL) kan bestemmes i henhold til den internationale standard ISO 1999:1990. Desværre er gyldigheden af metoden, til korrekt at kunne forudsige risikoen for høretab af alle typer af støj, stadig ikke verificeret.

Et kontroversielt aspekt omhandler støj af impulsiv karakter. Den nuværende opfattelse er, at risikoen for NIHL som følge af alle former for eksisterende støj i industrielle miljøer kan estimeres ud fra en energi basis. Derfor er der indført en standardmetode til vurdering af alle typer af støj, inklusiv impulsiv støj. Disse målinger er baseret på den samlede A-vgtede lydenergi af støjen i varigheden af eksponeringen ( $L_{Aeq,T}$ ), som er normaliseret til en normal arbejdsdag på otte timer  $L_{EX,8h}$ . Flere undersøgelser har vist, at for lige store mængder af energi, impulsstøj kan være mere farlig for vores hørelse end kontinuerlig støj. Af den grund tillader ISO 1999:1990 at tilføje en +5 dB straf, når der måles lyde af impulsiv karakter baseret på den formodning at den impulsive lyd udgør en større farer for høretab. Ikke desto mindre er sanktionen en forebyggende foranstaltning, da der i øjeblikket ikke foreligger tilstrækkelig systematiske data til at påvise +5 dB straffens gyldighed. Eller til at afgøre, om alle typer af impuls støj skal straffes.

Evalueringen af hørelsen og diagnosticeringen af høretab bliver typisk udført med den standardiserede ren-tone audiometri. Men den seneste indførelse af otoakustiske emissioner (OAE) kan give en mere følsom diagnosticering til evaluering af risikoen for NIHL. Forløbsundersøgelser og tværsnits studier har vist at OAE kan formindske fr audiogrammet hos grupper der er udsat for høje støjniveauer - hvilket antyder at ændringer i karakteren af det indre øre kan være prækliniske indikatorer på NIHL. Desværre er, vores forståelse af OAE mangelfuld, og forskningen er stadig igang med at undersøge, hvordan man kan optimere OAE målinger. Hensigten er at give de bedst mulige oplysninger om den sunde funktion af det indre øre og dens sårbarhed over for støj.

I den foreliggende afhandling blev, 16 normalt hørende forsøgspersoner, udsat for moderat støj-stimuli under laboratorieforhold ved to forskellige forsøg. Effekten af støj-stimulien på hørelsen hos forsøgspersonen blev overvåget med målinger af *distortion-product otoacoustic emissions* (DPOAEs). Eksperimenterne var begrænset til at evaluere midlertidige ændringer i karakteristikaene af DPOAEs - og var derfor reversible. De underliggende mekanismer mellem midlertidige og permanente auditive forandringer er sandsynligvis fysiologisk forskellige. Derfor kan vores resultater ikke ekstrapoleres til vedvarende eksponering i virkelige industrielle

miljøer. Ikke desto mindre, kan tilstedeværelse af en høj midlertidig forandring i en af stimuliene være en indikator på en højere risiko for NIHL.

Forsøgene havde følgende formål:

1. At undersøge, om antagelsen af at impulsiv industriel støj kan udgøre en større risiko for høretab end kontinuerlig støj med samme energiniveau. Forsøgspersonerne blev i 10 minutter udsat for to typer af binaural optagelser: (1) en kontinuerlig bredt båndet støj normaliseret til  $L_{EX,8h} = 80$  dBA, og en (2) kombination af den tidligere stimulus med støj af impulsiv karakter normaliseret til  $L_{EX,8h} = 75 + 5dB_{straf} = 80$  dBA. (peak niveau 117 dBC og en gentagelsesrate på 0.5 slag pr sekund). DPOAEs blev målt i et bredt frekvens område før og i de efterfølgende 90 minutter efter eksponeringen. Antagelsen er, at hvis straffen er korrekt, vil begge stimuli producere den samme effekt på DPOAEs.
2. At undersøge de metodologiske aspekter af DPOAE målinger, som kunne forbedre diagnoseringen og påvisningen af høretab. Især blev det undersøgt, om finstrukturen i DPOAE bliver systematisk påvirket efter en tonal overeksponering, og om evalueringen af finstrukturen kan give flere oplysninger om sårbarheden i det indre øre end på DPOAE niveauet alene. Forsøgspersonerne blev i 10 minutter udsat for 2 kHz tone normaliseret til et eksponeringsniveau  $L_{EX,8h} = 80$  dBA. DPOAE blev målt før og i de efterfølgende 70 minutter efter eksponeringen. Den eksperimentelle protokol tillod målinger med med en høj tids- og frekvensopløsning i en 1/3 oktavbånd centreret til 3 kHz.

Resultaterne indikerer, at:

1. +5 dB straffen overvurderede effekten på DPOAE niveauerne, og resultaterne kan bedre sammenlignes i forhold til deres totale akustiske energi. Det er i overensstemmelse med den nuværende opfattelse af, at risikoen for høretab fra lav niveau impulser, kan forudsiges på samme energi basis.
2. Finstrukturen i DPOAE er meget individuel, og ingen systematiske ændringer blev observeret efter støj overeksponering. Derfor er evalueringen af finstrukturen måske ikke en bedre indikator for risikoen for høretab. Tværtimod kan forskellene på tværs af forsøgspersonerne komplicere fortolkningen og sammenligningen af resultaterne.

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# Chapter 1

## Introduction

Noise-induced hearing loss (NIHL) is a permanent hearing impairment resulting from prolonged exposure to high levels of noise. In this way, NIHL is cumulative over a lifetime and it is not reversible by any presently available medical or surgical treatment. The degree of hearing loss can range from mild to profound and may also result in tinnitus. Hearing impairment has a major impact on one's communication ability and even mild impairment may adversely affect the quality of life. Yet NIHL is in theory preventable. Unfortunately, our modern lifestyle places many people at risk as we are often exposed to high levels of noise; either wanted –e.g., music concerts and portable music players– or unwanted –e.g., industrial machinery and traffic noise.

When the noise exposures occur at the workplace they are formally known as *occupational noise exposures*, and they constitute the most common source of NIHL. The World Health Organization reported in 2004 that NIHL from occupational exposures is still one of the most prominent and most recognized occupational disease in all regions of the world (Concha-Barrientos *et al.*, 2004). Both in United States and Europe 30 million people are exposed to potentially hazardous levels of noise, and worldwide the numbers are from 400 to 500 million. In addition, the *European Agency for Safety and Health at Work* estimates that the cost of hearing loss from noise in Europe represents about 10% of total compensation cost for occupational diseases (Schneider *et al.*, 2005).

The risk of NIHL is generally predicted according to the available noise standards (ISO 1999:1990; American National Standard ANSI S3.44-1996; OSHA 29 CFR 1910.95; NIOSH Publication No. 98-126). The standards are based on the current scientific knowledge regarding the functioning of the auditory system and its vulnerability to noise overexposure. Unfortunately, current scientific knowledge is insufficient to predict individual risk of hearing loss to a given exposure. For this reason, the standards establish a statistical method to estimate the percentage of people that might develop a hearing loss due to prolonged exposures to noises of different levels.

The current conception is that NIHL is a function of the total acoustical energy that reaches the inner ear throughout its life time. In this way, the standards propose a unified measurement method based on readings of the equivalent A-weighted level  $L_{Aeq}$ , and duration of the exposure within a normal working day of eight hours  $L_{EX,8h}$ . The method is based on the *equal-energy hypothesis* (EEH), which postulates that (1) noise exposures with the same A-weighted equivalent level ( $L_{Aeq}$ ) may produce similar effects on our hearing independently of their temporal and spectral properties; and (2) hearing loss is proportional to the acoustic energy received by

the ear. Therefore, an exposure to a particular noise level for one hour will result in the same hearing loss as an exposure for two hours to a noise level which is 3 dB lower than the original level<sup>1</sup> (Burns and Robinson, 1970; Martin, 1976).

Several longitudinal studies with industrial workers have demonstrated that the EEH provides a reasonable estimate of the risk of hearing loss in most industrial environments with continuous-type noise, i.e., noise with negligibly small fluctuations of level within the period of observation (e.g. Passchier-Vermeer, 1968, 1977; Burns and Robinson, 1970; Johnson, 1973; Cohen *et al.*, 1972). In addition, an important advantage of the standardized method is its simplicity – i.e., the risk of hearing loss is assessed with readings of one single number ( $L_{EX,8h}$ ), which facilitates the interpretation and comparison of results. Another advantage is that measurements of A-weighted levels do not require neither complex nor expensive instrumentation.

## 1.1 Do we measure impulsive noise correctly?

Opposite to continuous noise, industrial noise can also be of impulsive character. Impulse noise is defined as a rapid, often large change in the instantaneous sound pressure over a short period of time, typically less than one second (NIOSH Publication No. 98-126)<sup>2</sup>.

Noise standards establish that the EEH can be applied to all types of occupational exposures without hearing protection; also those of impulsive character as long as the C-weighted peak level does not exceed 140 dBC<sup>3</sup>. In this way, the assessment of occupational noise exposures becomes greatly simplified. However, it is still not clear whether the EEH can correctly predict the risk of hearing loss from impulsive exposures. The few available demographic field-studies investigating the effects of impulsive noise on the hearing of industrial workers have shown conflicting results, both in favor and against the applicability of the EEH for impulsive noises. On the one hand, there are studies that reported that the hearing losses of workers exposed to noise levels with impulsive components were higher than predicted according to the EEH. These studies were conducted with workers in steel construction (Passchier-Vermeer, 1971); building construction (Voigt *et al.*, 1980); drop-forge industry (Sulkowski and Lipowczan, 1982; Surovov *et al.*, 2001); and an automobile manufacturing plant (Thiery and Meyer-Bisch, 1988).

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<sup>1</sup>This is known as the “3-dB exchange rate” and it is widely accepted internationally. The 3-dB exchange rate is the method most firmly supported by the scientific evidence for assessing hearing impairment as a function of noise level and duration. However, some countries may use a more permissive exchange rate of 5 dB depending on their legislation. One of the arguments in favor of the 5-dB exchange rate is that noise exposures in many industrial environments are interrupted, i.e., there are resting periods in between exposures, which may allow the hearing of the workers to recover partially or completely. Although an exchange rate of 3-dB would be overprotective in truly intermittent situations, a 5-dB exchange rate would be underprotective in most others. For this reason, some noise standards like NIOSH Publication No. 98-126 have stopped using the 5-dB exchange rate and adopted the 3-dB rule.

<sup>2</sup>The term impulse (or impulsive) refers to the time characteristics of the acoustic signal. Further, impulsive noises can also be classified according to their generation mechanism as *explosive noise* or *impact noise*. Explosive noise is defined as a rapid expansion of gas, such as from discharges of weapons or explosions. On the other hand, *impact noise* is generated by the collision of two or more solid objects, like hammering.

<sup>3</sup>Noise standards adopted the use of C-weighted peak values in order to specify the frequency response of the instrument and eliminate very low frequency impulses and sounds. Without specifying the low end cutoff frequency of the instrumentation, measurements with different devices could vary greatly (Kardous *et al.*, 2005).



On the other hand, there are studies in the same industrial areas that reported that the hearing loss of workers exposed to impulsive noises could be predicted according to the EEH (Atherley and Martin, 1971; Guberan *et al.*, 1971; Taylor *et al.*, 1984). These contradictory results suggest that, up to certain limits, the EEH can be equally applied to impulsive and non-impulsive noises. Unfortunately, we simply do not have enough systematic data to delineate the range of conditions where the EEH is appropriate.

Another uncertainty regarding the effects of impulsive sounds on hearing refers to the combination of impulsive and continuous noise. In most industrial settings impulsive noise can occur simultaneously with other types of continuous background noise. Some researchers have suggested that this combination may have a synergistic effect in the inner ear. This is, the interaction of the two of them may pose a higher hazard to hearing than the sum of their individual contributions. However, this hypothesis is based on animal studies in which chinchillas were exposed to noises exceeding the properties of typical industrial settings – both in intensity and duration (Hamernik *et al.*, 1974; Hamernik and Henderson, 1976). When the exposure levels are comparable with those found in many common industrial environments the synergism seems to disappear (Hamernik *et al.*, 1981). Currently, whether the effects of combined exposures are additive or synergistic is still unknown.

### 1.1.1 The +5 dB penalty for impulsiveness

ISO 1999:1990 prescribes the same assessment method for impulsive and non-impulsive noises up to 140 dBC. Nevertheless, the standard allows adding a +5 dB penalty to the measured  $L_{Aeq}$  if a noise is of impulsive character based on the presumption that it might pose a higher risk of hearing loss. In this way, the standard considers that the risk of NIHL of such an impulsive exposure may be comparable to that from a continuous-type noise which is +5 dB higher in level.

The penalty is based on the available demographic studies showing that the hearing levels of workers exposed to widely fluctuating noises developed significantly larger losses (approximately 5 dB higher at 4 kHz) than workers exposed to more steady levels (Passchier-Vermeer, 1968; Taylor and Pelmear, 1976; Voigt *et al.*, 1980). For this reason, ISO 1999:1990 states: “The prediction method presented is based primarily on data collected with essentially broad-band steady non-tonal noise. The application of the data base to tonal or impulsive/impact noise represents the best available extrapolation. Some users may, however, want to consider tonal noise and/or impulsive/impact noise about as harmful as a steady non-tonal noise that is approximately 5 dBA higher in level.”

ISO 1999:1990 establishes that the penalty can be applied not only to impulsive noises but also to sounds with a prominent tonal component. However, ISO 1999:1990 does not specify when to penalize a given noise, neither for impulsiveness nor for tonality and the decision is left to the criteria of the observer. This uncertainty could lead to an overestimation of the auditory hazard, leading to excessively conservative legislation; or to an underestimation of the auditory hazard resulting in greater hearing loss on the exposed population.

The 5 dB penalty is a precautionary measure and there is not yet enough empirical data to validate the penalty. In fact, the maximum penalty for impulsiveness can vary up to 7 dB between countries depending on their noise legislation. Some countries like Denmark provide more specific guidelines regarding the applicability



of the penalty. Thus, the Danish Working Environment Authority<sup>4</sup> establishes that a +5dB penalty must be added when measuring noises of impulsive character with peak levels higher than 115 dBC (or dBA) more than once per minute (Arbejdstilsynet, 2003). It also establishes that the +5 dB penalty can be applied to noises with a clear tonal component. However, no precise guidelines are given in this case.

### 1.1.2 Needs of future research

Since 1960, extensive research mostly from animal studies has investigated those characteristics of impulsive exposures that may be hazardous for our hearing (for a review of the studies see Henderson and Hamernik, 1986). In addition, several investigators have proposed new exposure metrics to better quantify impulsive exposures in relation to their risk of hearing loss (Price, 2007; Hamernik *et al.*, 2003; Seixas *et al.*, 2005b; Stark and Pekkarinen, 1987, see section 2.1). However, there is not yet any clear defensible evidence to reject the current assessment methods established by the noise standards; nor to separate impulsive and continuous noise from occupational environments –and their effects– as long as the C-weighted peak level does not exceed approximately 140 dB (Von Gierke *et al.*, 1982). Peak levels above this value may damage our hearing instantly, and the effects cannot be predicted with the EEH. This level is known as the *critical level* and it is further explained in section 2.1.1.

The debate regarding the correct assessment of impulsive noise is still open, and it is internationally agreed among experts that future research should consider the following issues (Kardous *et al.*, 2005):

- Instruments and standards for measurement and evaluation of impulsive sounds must be revised.
- An international consensus on a damage-risk criterion for impulsive sounds is needed.
- More studies should be performed with an emphasis on either field surveys of exposure levels at the workplace and audiological data collection from human subjects.
- It is necessary to reach an international consensus on descriptors for impulsive sounds and procedures for applying results from tests on animals to models for the effect of impulsive sounds on hearing impairment of humans.
- It is necessary to examine the destructive or metabolic effects of impulsive sounds on the inner ear.
- It is necessary to investigate whether the combination of impulsive and continuous noise might have a synergistic effect on the auditory system.

## 1.2 Importance of otoacoustic emissions in the assessment of NIHL

Several methods exist to assess hearing function and detect NIHL. The most common is the traditional pure-tone audiometry consisting of estimations of hearing

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<sup>4</sup>[www.arbejdstilsynet.dk](http://www.arbejdstilsynet.dk)

thresholds; i.e., the minimum sound level that a person can hear at given frequencies. However, otoacoustic emissions (OAEs) may provide a better diagnosis tool to study the vulnerability of the inner ear to noise overexposure, and to detect and prevent early hearing-loss.

OAEs reflect the status of the cochlea in the inner ear. It is commonly accepted that OAEs are a sensitive indicator of the physiological activity of the outer hair-cells (OHCs) in the cochlea. The role of the OHCs is essential for a healthy hearing. Unfortunately, they are also the most vulnerable part of the ear in response to noise and it is commonly agreed that the onset and gradual development of NIHL is mainly a consequence of OHC loss (Saunders *et al.*, 1991). For this reason OAEs are extremely valuable in the assessment of NIHL – because OAEs reflect vulnerability of the inner ear after acoustic overexposure due to metabolic changes in the OHCs (Kemp, 1986; Probst *et al.*, 1991). Besides, OAEs are objective –i.e., no active response from the subject is necessary for their assessment. This is a significant advantage over behavioral audiometry, as for measurements of hearing thresholds the concentration of the subject may be a critical factor influencing the variability of the results.

Most of what is known about the effect of long-term occupational noise exposures on the human auditory system is based on audiometric measurements evaluating temporary (TTS) or permanent hearing-threshold shifts (PTS). Some of these studies are also the basis of the current noise legislation (e.g. Passchier-Vermeer, 1968, 1977; Burns and Robinson, 1970; Johnson, 1973). However, longitudinal and cross-sectional studies have shown that OAEs can diminish before hearing levels in populations exposed to high levels of noise (LePage and Murray, 1993; LePage *et al.*, 1993; Murray and LePage, 1993; Miller *et al.*, 2006; Hamernik and Qiu, 2000; Prasher and Sulkowski, 1999; Marshall *et al.*, 2009; Attias *et al.*, 2001; Engdahl *et al.*, 1996; Seixas *et al.*, 2005a; Miller *et al.*, 2004; Konopka *et al.*, 2005; Attias *et al.*, 1995). From this studies it is generally accepted that OAEs may be more sensitive than traditional pure-tone audiometries to detect incipient NIHL. One of the reasons for the purported added sensitivity of OAEs refers to OHC redundancy. There is physiological evidence for OHC redundancy in animals, such that only some OHCs are required for normal hearing (e.g., Altschuler, 1992). This redundancy provides some protection from noise-induced damage, and it is only when critical number of OHCs are damaged that hearing suffers. LePage *et al.* (1993) suggested that because OAEs are a direct consequence of OHC activity, the loss of some OHCs is likely to be measurable with OAEs before being measurable with an audiogram. In this way, the OHC redundancy theory suggests that changes in OAEs could be preclinical indicators of NIHL.

Currently, OAEs cannot replace traditional pure-tone audiometries to diagnose NIHL, and the audiogram is still considered the *gold standard*, to which OAE measurements are compared. Rather, OAEs are a complementary measurement. One of the reasons for this is that our understanding of OAEs is not complete and researchers are still investigating how to optimize OAE measurements in order to provide the maximum information regarding the healthy functioning of the inner ear and its vulnerability. At present, there is no standardized method regulating the use of OAEs for the detection of NIHL and researchers from all over the world might be using different measurement protocols in their investigations. This inconsistency makes very difficult the comparison and interpretation of results from different studies.

The literature regarding the effect of impulsive sounds on the OAEs of humans

is still sparse and it has mostly focused on military settings (Marshall *et al.*, 2001; Attias *et al.*, 2001; Engdahl *et al.*, 1996; Konopka *et al.*, 2005; Miller *et al.*, 2004). In these studies, OAEs were used to detect incipient hearing loss in military personnel exposed to impulsive noise from fire weapons and artillery. Changes in the properties of the OAEs were detected, whereas no change in hearing thresholds was observed (see section 2.3.5). Results from these studies are extremely valuable –as they help us to understand how PTS develops in humans. However, they also have some limitations. One limitation is that it is difficult to control all the variables in the experiment and it is not possible to perform measurements comparable in quality to those made in the laboratory. Thus, it is very difficult to understand from these studies how the different characteristics of impulsive sounds may influence the risk of NIHL. Another limitation is that the results cannot be extrapolated to industrial environments because peak levels from weapons are generally above 140 dBC, and hence, beyond the scope of ISO 1999:1990.

To our knowledge, there are no controlled laboratory-studies that have systematically investigated the effects of industrial impulsive noise on the OAEs of human subjects. This type of data may provide a valuable insight into the hazardous properties of impulsive noise on our hearing. Laboratory studies on humans are restricted to evaluate temporary changes on the auditory system. The underlying mechanisms between temporary and permanent shifts are likely to be physiologically different, both for hearing thresholds (Saunders *et al.*, 1985) and OAEs (Lapsley Miller and Marshall, 2006). In this sense, TTS and PTS appear to be produced by different mechanisms (Nordmann *et al.*, 2000) with TTS due to inactivation of mechano-electrical transduction (MET) channels at the apex of OHCs (Patuzzi, 1998) while PTS involves hair cell death due to free radicals (Samson *et al.*, 2008). For this reason, results from laboratory studies cannot be expected to generalize to prolonged exposures from real-life situations. However, the presence of a temporal shift may be understood as a warning sign, indicating that there might be a risk of a permanent hearing loss for prolonged exposures.

Two types of OAEs have been extensively used for the assessment of NIHL: transient-evoked otoacoustic emissions (TEOAEs) and distortion-product otoacoustic emissions (DPOAEs). In the present study DPOAEs are chosen for the evaluation of changes in the inner ear of human subjects. This choice is argued in section 2.3.6.

### 1.3 Goal of the study

The present dissertation investigates the effect of acoustic overexposure on the DPOAEs of human subjects. Two different experiments were performed. The experiments are presented in detail in two different journal papers in pages 87 and 97 respectively. In the following, the experiments are referred to as PAPER I and II and they had the following purposes:

PAPER I: *Recovery of distortion product otoacoustic emissions after a 2-kHz monaural sound-exposure in humans: effects on fine structures*

To investigate methodological aspects of DPOAE measurements that might improve hearing diagnosis and detection of hearing loss. In particular, it is studied whether the fine structures of the DPOAE (explained in section 2.3.3) are systematically affected after a moderate tonal overexposure, and whether the evaluation of

the characteristics of the fine structures might provide more information about the vulnerability of the cochlea and detection of incipient hearing loss.

PAPER II: *Is it necessary to penalize impulsive noise +5 dB due to higher risk of hearing damage?*

To investigate the validity of the +5 dB for impulsiveness established by ISO 1999:1990 for predicting risk of NIHL. Human subjects were exposed under laboratory conditions to two different noise stimuli. One of the stimuli was penalized +5 dB for impulsiveness as specified by the Danish Working Environment Authority (Arbejdstilsynet, 2003). The purpose was to investigate whether there is a difference in the temporary changes from the two stimuli, which may be indicative of a higher risk of NIHL. The assumption is that, if the penalty is correct, both stimuli may produce a similar effect on the DPOAEs.

It is also the purpose of both experiments to provide human data reflecting the auditory hazard from overexposures to different noise stimuli and their effects on DPOAEs. This is particularly important in the case of impulsive noise, as most of the literature regarding the effects of impulsive noise on hearing is based on animal studies, and the results can not be extrapolated directly to humans.

The rest of the present dissertation is organized as follows: chapter 2 presents the most relevant theory setting the scientific basis of our investigation. A description of the methods, i.e., selection of noise stimuli and OAE assessment, are presented in chapter 3. The main results are shown in chapter 4. For a more detailed analysis and discussion of individual data, please refer to the corresponding papers. A general discussion can be found in chapter 5.

The methodological protocol for the assessment of DPOAE is explained in a conference paper in page 109.



# Chapter 2

## Background and theory

This chapter presents the most relevant theory establishing the scientific basis for this thesis. The chapter starts with a review of the current knowledge regarding risk of hearing damage from impulsive noise. It is explained why impulsive noise may be more dangerous for our hearing than non-impulsive noise as well as current alternate metrics for the assessment of impulsive noise exposures. Next, the chapter continues with a description of OAEs and their validity to assess the vulnerability of the cochlea to noise overexposure.

### 2.1 Auditory effects of impulsive noise

High-intensity impulsive sounds are considered to be more dangerous to hearing than continuous sounds (Henderson and Hamernik, 1986; Stark *et al.*, 2003). One explanation is that the auditory system may respond differently to impulsive noise than to continuous noise.

First, the stapedius-reflex muscle in the middle ear provides no protection against a sudden impulsive sound. The stapedius reflex attenuates loud sounds before they reach the cochlea, and thereby, the risk of noise-induced hearing loss is reduced. For pure tones, sound levels above 75 dB HL will activate the reflex. The reflex protects mainly against sounds in the low frequency range, whilst sounds above 2 kHz are negligibly affected. At maximum contraction of the reflex, the attenuation provided is around 20dB at low frequencies. The contraction of the muscle does not appear immediately when a loud sound occurs, but with a latency of at least 10-20 ms. For this reason, impulsive noises may escape the protective effect of the reflex, and reach the cochlea without attenuation (Arlinger, 1993).

Secondly, the spectral energy of an impulse is often broadly distributed across a wide range of frequencies. Therefore, high-level impulsive noise may have a direct damaging impact over a broad region of the basilar membrane (Dunn *et al.*, 1991).

The physical properties of an impulse are characterized by the peak level, rise and decay time, repetition rate of the impulse, number of impulses and spectral content. Several studies have indicated that these parameters might be critical in the development of hearing loss (for a review see Henderson and Hamernik, 1986). Understanding the effects of the aforementioned parameters on the auditory system is complicated as the parameters can vary over a very wide range. Besides, it is very difficult to study the influence of individual parameters in laboratory experiments because the manipulation of one parameter may also influence the others. For example, a variation in the decay time of an impulse may affect the energy content in

a particular frequency range. Next, the main parameters characterizing an impulse and their influence in the development of NIHL are presented.

### 2.1.1 Peak level

The peak pressure-level is one of the key parameters in the development of NIHL. The current conception is that the higher the peak level, the higher the risk of damage. Unfortunately, there is not a simple relationship between the amplitude of an impulse and either cochlear damage or hearing loss. Currently, it is believed that there might be two different mechanisms of damage depending on the intensity of the peak level. The mechanisms are classified as *metabolic change* and *mechanical damage*. An excellent review and tutorial regarding the two mechanisms and anatomical injuries in the inner ear after acoustic overexposure is given by Saunders *et al.* (1991, 1985); Henderson and Hamernik (1986) and Schmiedt (1984). Next, the most important findings are briefly presented.

#### Metabolic changes

Metabolic changes have a gradual onset and they refer to those changes that affect the vital functions of the outer and inner hair cells (OHC and IHC). The changes may be temporal for moderate exposures. Temporal changes are not completely understood but may include vascular changes, metabolic exhaustion, and chemical changes within the hair cells. There is also evidence of a decrease in the stiffness of the stereocilia (the hair bundles at the top of the hair cells). This decrease in stereocilia stiffness may lead to a decrease in the coupling of sound energy to the hair cells, which thereby alters hearing sensitivity (Cody and Russell, 1986).

Repeated exposure to noise may gradually cause permanent NIHL. In this type of injury, a few scattered OHC may be damaged with each exposure. With continued exposure, the number of damaged hair cells increases. Damage to the stereocilia is often the first change. Once destroyed, the sensory cells are not replaced. For prolonged intense-exposures the degeneration of hair cells may continue. Once a sufficient number of hair cells are lost, the nerve fibers of that region also degenerate. The extent to which these neural changes contribute to NIHL is not clear (Hamernik *et al.*, 1993, 1989).

#### Mechanical damage

Opposite to metabolic changes, mechanical damage may occur instantly. Mechanical damage is irreversible and it might lead to direct PTS. Virtually all the structures of the ear can be damaged, in particular the organ of Corti. Typical mechanical damage includes dislocation of the ossicles, rupture of the tympanic membrane, destruction of OHC/IHCs and changes in the morphological properties of the inner ear, among others (Saunders *et al.*, 1991).

#### The critical level

According to literature, there is a certain peak level that marks the transition between *metabolic changes* and direct *mechanical damage* in the cochlea (Ahroon *et al.*, 1993; Qiu *et al.*, 1986; Roberto *et al.*, 1985). This level is known as *the critical level*.



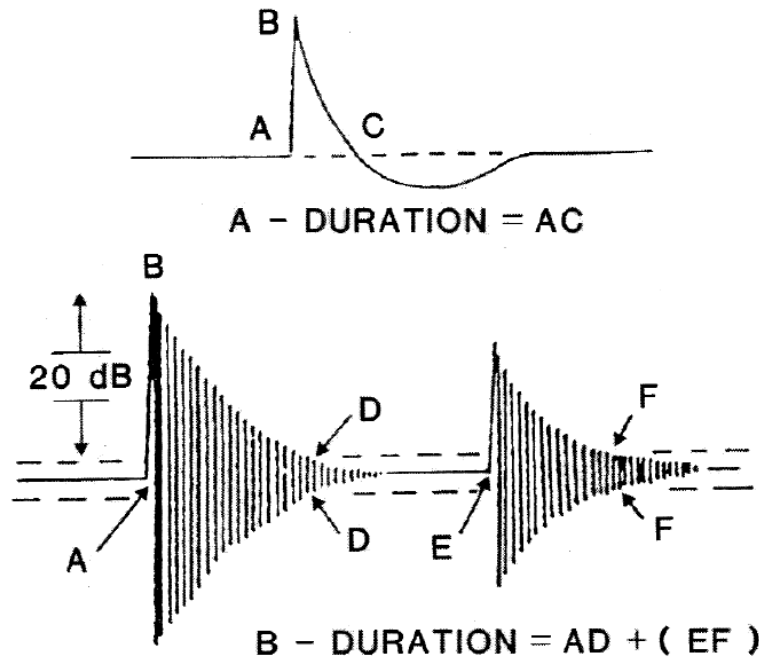


Figure 2.1: Conceptual representation of the A and B-duration. Taken from Stark *et al.* (2003).

Henderson *et al.* (1991) measured TTS and PTS in groups of chinchillas exposed to different equal-energy stimuli. The stimuli consisted of series of broad-band impulse noise with peak levels from 107 to 137 dB SPL. The authors found great variability in the results and concluded that the critical level for chinchillas may be between 119 and 125 dB. The value of the critical level has never been verified for humans. However, if a 20-dB adjustment is used to account for the difference in susceptibility between chinchillas and humans (Ahroon *et al.*, 1993), the critical level extrapolated for humans may be between 139 and 145 dB –although it commonly appears as 140 dBC in the literature (NIOSH Publication No. 98-126, 1998). Further, the dependence of the critical level with the frequency content of the impulse is not known.

It is commonly accepted that peak levels below the critical level may conform the EEH principle. On the contrary, impulsive sounds above the critical level may result in an immediate, severe and permanent hearing loss by causing a direct mechanical damage in the cochlea, also termed as *acoustic trauma*. Stimulation above the critical level results in a rapid growth of damage, which might be severely increased for prolonged exposures (Roberto *et al.*, 1985).

### 2.1.2 A and B-duration

The A-duration is defined as the time from the onset of the impulse to the first zero level crossing. The B-duration is defined as the time during which the envelope of pressure fluctuation stays within 20 dB of the peak SPL, also including the recurrent impulses. Both A and B-duration are depicted in Figure 2.1.

It is considered that for industrial settings, in which peak levels are generally below 140 dBC, the B-duration might be a better descriptor of the impulse. This is because impulse noise from industrial settings is typically reverberant and for this reason, the B-duration may include all the reflections from nearby sources. The



A-duration, on the other hand, is considered more adequate for the characterization of high-level impulse sounds in free field, like those typically present in the military.

The number of experiments studying the influence of the B-duration on the development of hearing loss is sparse. Trémolirès and Héту (1980) and Yakamura *et al.* (1980) investigated in similar experiments the influence of the B-duration on the development of TTS in human subjects. Basically, both experiments exposed subjects to noise stimuli with the same peak level but different B-duration, while measuring the growth of TTS at particular frequencies. They concluded that the longer the B-duration of the impulse, the higher the risk of hearing loss. However, the following observations can be made to their conclusion:

1. Manipulating the B-duration of an impulse may also have an effect on its spectral properties. Therefore, the higher TTS measured at a particular frequency may have been due to a higher energy content in that frequency range.
2. Even though noise exposures had the same peak level, a longer B-duration implies more acoustic energy. Therefore, perhaps the differences in the measured TTS could have also been predicted according to the EEH principle.

### 2.1.3 Rise time

The rise time is defined as the time, in seconds, that an impulse takes to rise from 10% to 90% of its maximum absolute value of the sound pressure.

There are almost no studies that have systematically investigated the hazardous properties of the rise time of occupational impulsive noise on humans. This type of data is difficult to collect due to technical limitations of the reproduction system, the loudspeaker being the weakest element. Thus, it is very difficult to have an accurate reproduction and control of a given rise time for high-level peaks. To our knowledge the only available study is the one by Trémolirès and Héту (1980). They reported that the rise time did not appear to be a relevant factor in the development of hearing loss. Their conclusion is based on the fact that three different impulsive stimuli produced a similar amount of TTS on the hearing of the subjects, despite having a different rise time.

### 2.1.4 Repetition rate

Several human and animal studies have shown that for equal energy exposures the repetition rate might be a critical factor in the development of hearing loss (Trémolirès and Héту, 1980; Danielson *et al.*, 1991; Henderson *et al.*, 1991). The literature suggests that this effect is due to two important factors that vary with time: the acoustic reflex of the middle ear and the rate of recovery following an impulse. The current conception is that a series of impulses with a repetition rate of approximately one impact per second is the most dangerous for our hearing. This is because impulses within this range are not attenuated by the middle-ear reflex, and pose the highest rate of acoustic stress to the inner ear.

### 2.1.5 Number of impulses

The Committee on Hearing, Bioacoustics, and Biomechanics (CHABA) of the U. S. National Research Council proposed the first damage-risk criterion (DRC) in 1968 for

impulsive noise (CHABA, 1968). This DRC considered the total number of impulses within an exposure a critical parameter in the development of hearing loss. The CHABA criterion allowed 100 impulses per day at 140 dBA, or 1000 per day at 130 dBA, or 10,000 per day at 120 dBA. However, these limits were more an “educated guess” based on the relatively few experimental results and observations that were available at that time. For this reason, the CHABA criterion is no longer valid and it is recognized now that the CHABA criterion was inadequate and oversimplified.

Currently, there are no limitations regarding the maximum number of impulses that a person can be exposed to within a normal working day of eight hours, as long as they do not exceed a certain peak level. ISO 1999:1990 establishes a maximum peak level of 140 dBC based on the concept of the critical level. However, some countries may have more restrictive limits based on their legislation. In France, for example, the maximum peak level is set to 135 dBA.

### 2.1.6 Spectral content

For impulses with peak levels above the critical level, the spectral content of the impulse seems to be crucial for the frequency specificity of hearing loss. Patterson *et al.* (1993) exposed groups of chinchillas to different types of narrow-band impulses containing the same energy but concentrated at different frequencies. Peak levels were above the estimated critical level of the chinchilla. Their results showed that hearing hazard based on estimations of PTS is closely related to the spectral content of the impulse. Besides, low-frequency impulses were less dangerous than impulses with mid-range frequencies. The authors concluded that low-frequency impulses are less hazardous than predicted by A-weighted sound exposure level. In addition, audiometric data from military settings have shown that the hazard to hearing posed by exposure to impulse noise is strongly influenced by the spectral distribution of the energy of the impulse (NATO, 1987; CHABA, 1992).

In industrial environments, whether the spectral content of impulsive noise is critical for the development of hearing loss is still not clear. Brüel (1977) stressed that current noise standards -based on measurements of A-weighted levels- may be inaccurate to account for the spectral differences of impulsive and continuous noise. The main concern is that the A-weighting is an approximation of the equal-loudness contour at 40 dB above threshold. This fact, per se, introduces two conflicting issues: (1) at higher levels the equal-loudness contours become flatter; and (2) the equal-loudness contours are based on loudness estimation of pure tones. Therefore, the A-weighting may not be accurate to describe the frequency-dependent sensitivity of our hearing for high-level impulsive sounds. Nor to account for their risk of hearing damage. Brüel (1977) also emphasized that the majority of industrial noise has a higher intensity in the 250–500 Hz frequency range, whereas short duration peaks contain a significant amount of energy in the 4–6 kHz frequency region. Because the frequencies in the 4–6 kHz are also amplified in the outer and middle ear, the short duration peaks may play a dominant role in contributing preferential damage in this range.

Tambs *et al.* (2006) compared the frequency specificity of NIHL across the range 250–8000 Hz and the extent to which the patterns of frequency-specific auditory threshold shifts differ between occupational noise and impulse noise from shooting. A total of 51975 subjects participated in the study. Subjects underwent a pure-tone audiometry together with a questionnaire about their noise exposure history and

other risk factors. Data from pure-tone audiometries were classified in groups according to age, gender, type and duration of exposure. The results indicated that for men above 65 years, the effects of impulsive noise were strongest at 3000–8000 Hz and varied little within this frequency range, with a maximum shift of approximately 8 dB; whereas occupational noise had a higher impact at 3000–4000 Hz with a 13 dB shift at 3000Hz. These results suggest that high-peak impulse noise may have a bigger impact on high frequencies than occupational noise in general. They also suggest that there is an overlapping range at 3000–4000 Hz in which the cochlea is more vulnerable to NIHL for all types of noise exposure. However, the following observations can be done to their study: first, the comparison of hearing loss from occupational noise and impulses from weapons is not straightforward, as their destructive mechanisms on the auditory system are essentially different. Second, occupational exposures also included sources of impact noises such as riveting and hammering, although at lower intensity levels than impulses from shooting. This fact may have biased the results and the overlapping frequency range at 3000–4000 Hz may be due to the common action of impulsive noises in that area.

The similarities in the frequency-specific hearing loss between continuous and impulsive noise have been reported by other longitudinal studies on workers from different industrial areas (Passchier-Vermeer, 1968; Ceypek *et al.*, 1973; Sulkowski and Lipowczan, 1982; Taylor *et al.*, 1984). From these studies, it is commonly agreed that the development of hearing loss seems to be more intense during the first 10 years of exposure, with the highest PTS between 4–6 kHz for both types of noise. However, it is still not clear whether the development of hearing loss is faster for impulsive sounds.

## 2.2 Alternate metrics for impulsive noise exposures

Several researchers have proposed alternate metrics to better evaluate the auditory hazard of impulsive exposures. These alternate metrics do not intend to replace the current assessment method proposed by the standards, but rather to complement it and provide a better insight in the evaluation of NIHL.

### 2.2.1 Cumulative crest factor

Stark and Pekkarinen (1987) proposed a statistical method to describe the impulsive characteristics of a noise. The method is known as the *cumulative crest factor* and it allows classification of noise environments into impulsive or non-impulsive. According to the method, a noise environment is considered impulsive when the difference between the peak and RMS levels of a sound pressure is equal to or greater than 15 dB. The expression is given by

$$L_{Ap} - L_{AS} \geq 15dB \quad (2.1)$$

The expression is simply an A-weighted crest factor, where  $L_{Ap}$  is the A-weighted peak level and  $L_{AS}$  is the A-weighted RMS-level measured with “slow” time constant. The numerical value of 15 dB was accepted in the resolution of the International Symposium on the Effects of Impulse Noise on Hearing (Lahti and Starck, 1980). This criterion value corresponds to the averaging time of the ear so that the

loudness perception of impulsive sounds matches the loudness of continuous sounds, and it is determined to be approximately 35 ms (Brüel, 1977).

The difference between  $L_{Ap}$  and  $L_{AS}$  varies randomly in industrial noise. Therefore, the method calculates the cumulative distribution function, which consists on calculations of the crest factor at small intervals of time. In this way, the method determines the percentage of time in the sample noise exceeding 15 dB. However, the method does not establish a fence value regarding the percentage of time that the crest factor must exceed 15 dB for a noise to be considered impulsive. Therefore, rather than indicating that the method allows classification of noise environments into impulsive/non-impulsive, it would be more precise to state that noise environments are classified from highly to barely impulsive.

Stark *et al.* (2003) studied whether the hearing loss developed by forest and shipyard workers could be predicted according to ISO 1999:1990. Both acoustic environments were very complex, with random incidences of impulses combined with background noise. They used the cumulative crest factor to study the degree of impulsiveness of the noise environments. They concluded that the hearing loss developed by forest workers in a non-impulsive environment could be predicted according to the standard; whereas shipyard workers in an impulsive environment developed a higher hearing loss that could not be predicted with the standard. The authors suggested that the cumulative crest factor might be an appropriate complementary method to identify hazardous impulsive environments that might lead to a higher hearing loss than predicted according to ISO 1999:1990. Similar results were obtained by Surovov *et al.* (2001) in a study with forge hammering workers.

Unfortunately, at present there is not enough data to illustrate how the risk of hearing loss may depend on the overall level of the noise and its crest factor. For this reason, the method is more used as a warning sign to indicate that if a noise environment has clear impulsive content, the risk of hearing loss may not be correctly predicted according to the EEH.

### 2.2.2 Kurtosis

Erdreich (1986) proposed the kurtosis metric ( $\beta$ ) as a statistical descriptor of impulsiveness. Basically, the kurtosis calculates the *peakedness* of a sound based on its amplitude distribution. The analysis is done by dividing the time-pressure sound signal in small analysis windows. The advantage of the kurtosis over other the cumulative crest factor is that all peaks are accounted for in the calculation and that the relative difference between peak and background level is also incorporated; while the cumulative crest factor only computes the maximum peak level in a given time frame.

The kurtosis is calculated as the ratio of the fourth moment of the amplitude distribution to the squared second moment of the distribution in the analysis window. Thus for a window with  $N$  samples from  $x_1$  to  $x_N$ ,

$$\text{fourth moment, } m_4 = \frac{1}{N} \sum_{i=1}^N (x_i - \bar{x})^4 \quad (2.2)$$

and

$$\text{second moment, } m_2 = \frac{1}{N} \sum_{i=1}^N (x_i - \bar{x})^2 \quad (2.3)$$

The kurtosis ( $\beta$ ) is given by,

$$\beta = m_4/m_2^2 \quad (2.4)$$

A pure continuous noise (Gaussian) will have a kurtosis value of  $\beta = 3$ ; while purely impulsive sounds (non-Gaussian) may reach values of  $\beta > 100$ .

Audiometric and histological<sup>1</sup> experiments with chinchillas have suggested that the kurtosis metric might be a better descriptor of risk of hearing loss for impulsive noise (Hamernik *et al.*, 2003, 1993). In these experiments groups of chinchillas were exposed to different types of noise from continuous to impulsive ( $3 < \beta < 105$ ) with equal energy. The noise stimuli were designed artificially so that they all had the same spectrum. The results indicated that permanent auditory effects of the exposures (PTS) were higher as  $\beta$  increased; i.e., as the noise stimulus became more impulsive, and the PTS reached an asymptote at  $\beta \approx 40$ . The authors concluded that the kurtosis might be an important variable to assess risk of hearing damage from industrial environments. They also emphasized the limitations of the EEH for the assessment of impulsive noises. However, the following observations can be made to their conclusions: (1) the noise exposures were very unrealistic compared to real-life situations, as they consisted of uninterrupted exposures during five days; and (2) noise stimuli had peak levels in the range of 104–130 dB. Considering that the critical level for the chinchilla is approximately 120 dB, the authors may have compared audiometric data from groups with mixed metabolic (and only metabolic) changes and metabolic+mechanical damage in the inner ear.

In the same manner as the cumulative crest factor, it is still not known how the degree of hearing loss may depend on the kurtosis of an impulsive signal and its overall exposure level.

### 2.2.3 The AHAH model

The *Auditory Hazard Assessment Algorithm for the Human* (AHAH) is a theoretically based mathematical model of the ear designed to predict hazard of high-level acoustic impulses using computer-simulation techniques (Price and Kalb, 1991; Price, 2007). The AHAH model was developed by the U. S. Army in order to predict risk of hearing loss from military weapons. Peak levels typically encountered in military settings can be in the range of 130–190 dB in free field, and the assessment of hearing hazard from these extreme exposures is full of difficulties for the following reasons:

- Technical limitations of the instrumentation for the assessment of high-intensity peak levels.
- Lack of knowledge about the destructive mechanisms of the inner ear from high-intensity sounds.
- Difficulties to correctly document the wide range of noise exposures in military settings and verify the validity of the existing damage-risk criteria (CHABA,

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<sup>1</sup>Histological: Referring to the microscopic structure of organic tissues.



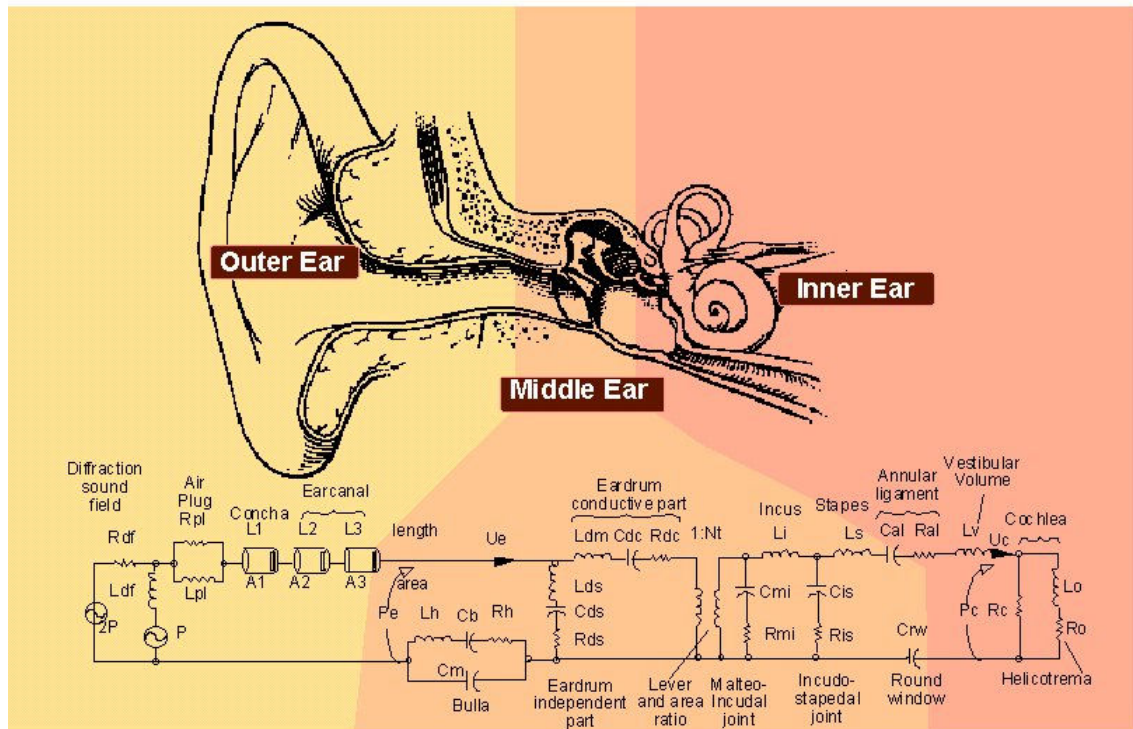


Figure 2.2: Electro-acoustic analog of the ear as defined by the AHAH model (Price and Kalb, 1991).

1968; Cheng *et al.*, 1987; MIL-STD 1474(B), 1979). Hence, the consensus of the scientific community is that none of them is accurate (Chan *et al.*, 2001; Kardous *et al.*, 2005).

The AHAH is essentially a theoretically based electro-acoustic analog of the ear designed to predict hazard from any intense sound, where the loss mechanisms within the inner ear are thought to be essentially instantaneous mechanical stress. The electro-acoustic analog of the ear is depicted in Figure 2.2. Briefly, the model calculates changes in the flow of energy from free field to basilar membrane displacement. The calculation is carried out at 23 locations evenly spaced along the basilar membrane (roughly 1/3 octave bands). At each location, the upward movement of the basilar membrane is tracked. Their amplitude in microns is squared and the sum is maintained for each location. The units are called Auditory Damage Units (ADUs).

A total of 500 ADUs is the maximum allowable dose in a single exposure. The model assumes that this number will correspond to a TTS of 25 dB, and that this is the maximum shift from which the ear can recover completely. Therefore, higher doses are predicted to produce a permanent hearing loss.

If the exposure consists of several impulses, the AHAH adds the contribution of each impulse in ADUs. Thus, one big impulse with 500 ADUs is equivalent to five impulses with 100 ADUs, or 100 impulses with 5 ADUs, etc. In this manner, the model estimates the maximum number of impulses that a subject can tolerate within a certain exposure without introducing a permanent hearing damage.

The initial development of the AHAH model was based on the cat ear, where TTS, PTS, and cellular changes could be examined (Price and Kalb, 1991). As mammalian cochleas are highly similar; the authors assumed that the loss processes that operate in the cat ear are likely to operate in the human ear. Only the co-

efficients in the equations were changed to reflect the differences in physical sizes between the two. Initially it was assumed that the model would have to be adjusted to improve the fit of the data in human ears, but the available studies with human volunteers have shown good correspondence between the hearing loss data and the model's predictions (Price, 2007). For this reason, the model has stayed in its original form.

The AHAAH model still needs to be validated with more human data from real-life situations. Nevertheless, there is a great expectation among the scientific community in the development of the AHAAH model as it has the potential to serve as an international design standard for weapons, and to provide damage or risk criteria for intense impulses of industrial origin (Kardous *et al.*, 2005). Recently, the AHAAH model has been incorporated by automotive companies to assess risk of hearing damage from airbags, which can reach peak levels up to 170 dB in the ear of the vehicle occupant (Rouhana *et al.*, 2005).

More information about the AHAAH as well as a free version of the software can be found at the official website of the U.S. Army<sup>2</sup>.

## 2.2.4 Applicability of AHAAH for occupational impulsive noises

It is not clear yet whether the AHAAH can be used to predict risk of hearing damage caused by impulsive sounds from occupational exposures (Kardous *et al.*, 2005). The main reason is that the model is intended to work with peak levels above the critical level, and to account mainly for mechanical changes in the inner ear; while peak levels from industrial environments are typically below the critical level and they are assumed to produce mainly metabolic changes. Therefore, the AHAAH model may not predict accurately hearing damage from occupational impulsive noises.

In the following, some of the limitations that the AHAAH introduces to account for metabolic changes in the inner ear are explained.

Although the AHAAH accounts for the effect of the acoustic reflex of the middle ear, it only considers two options: either ON, e.g., the muscle is fully contracted before the arrival of the impact; or OFF, the muscle is deactivated and it contracts reflexively in response to the impulse. For estimations of the hazard from repeated exposures to the same impulse, the model simply adds the hazard (in ADUs) of each individual exposure. While this might hold for the assessment of direct mechanical damage from high-intensity peak levels, it might not be true in the case of occupational exposures to a series of impulses. In this case the AHAAH model might overestimate the hazard for two reasons:

1. For repetitions of impulses the AHAAH does not consider that the reflex muscle may be partially activated when an impulse arrives; which might attenuate sound transmission to the inner ear and, therefore, result in a protective effect (Henderson and Hamernik, 1986; Arlinger, 1993).
2. The AHAAH does not consider that the temporary spacing between impulses may allow the ear some time to recover.

Another limitation is that the AHAAH estimates risk of mechanical damage based on measurements of the displacement of the basilar membrane in ADUs.

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<sup>2</sup><http://www.arl.army.mil>

However, it does not account for *metabolic exhaustion* of the outer hair cells (OHC), which could be a better indicator of risk of hearing loss from occupational exposures. Finally, it is not clear whether the model can predict risk of hearing loss from combinations of impulsive and continuous noise, as typically encountered in industrial environments. For these reasons, there is still some skepticism to use the AHAH for the assessment of occupational NIHL (Kardous *et al.*, 2005).

## 2.3 Otoacoustic emissions

Otoacoustic emissions (OAEs) are sounds generated by the inner ear as part of the normal hearing process. These sounds can be detected in the ears of mammals, including humans. OAEs can be recorded with a sensitive microphone fitted into the ear canal of the subject. In this way their assessment is objective, innocuous and non-intrusive.

It is believed that OAEs are a byproduct of the active amplification process within the cochlea (Kemp, 2002). During this process the OHCs enhance the vibration of narrow regions of the basilar membrane (BM), which improves low-level sensitivity and refines the frequency selectivity of the mechanical vibrations of the cochlea. A consequence of the process is that some of the acoustic energy travels backwards through the middle ear from the cochlea. This energy will make the tympanic membrane vibrate and, therefore, radiate sound into the ear canal in the form of OAEs. An unobstructed ear canal and a healthy middle ear are necessary for a correct detection of OAE levels. The OHCs are an essential part of this process. For this reason, it is commonly accepted that OAEs are a sensitive indicator of the physiological activity of the OHCs in the inner ear (Probst *et al.*, 1991).

### 2.3.1 OAE vs. hearing thresholds

The presence of OAE is interpreted as a sign of healthy hearing. This statement holds for population studies for which it has been shown that the presence of OAEs is related to normal hearing thresholds; whereas reduced OAE levels correlate to elevated hearing thresholds (e.g., Collet *et al.*, 1990; Desai *et al.*, 1999; Lonsbury-Martin *et al.*, 1991; Reuter and Hammershøi, 2007). For individual diagnosis OAE measurements might not be sufficient to infer that a subject has normal hearing.

Hitherto, it is not possible to predict hearing thresholds based on OAE measurements (e.g., Gaskill and Brown, 1990, 1993). One explanation is that OAE and hearing levels involve two different auditory mechanisms. On the one hand, OAEs do not provide information about the hearing beyond the cochlea; whereas hearing thresholds are the result of a cognitive process that depends not only on the cochlear status but also on the further processing. For this reason, OAE measurements cannot be considered a hearing test. Instead, OAEs are an objective test of cochlear function.

### 2.3.2 Types of OAE

OAEs can occur either spontaneously or in response to an acoustic stimulus. The former are called *spontaneous otoacoustic emissions* (SOAEs) and the latter *evoked otoacoustic emissions* (EOAEs).

SOAEs occur in the absence of any intentional stimulation of the ear. The presence of SOAEs is usually considered to be a sign of cochlear health, but the



absence of SOAEs is not necessarily a sign of abnormality. In fact, their prevalence is about 40-60% of normal hearing people and for this reason, SOAEs have little or none clinical application.

There are several techniques for the analysis of EOAEs. The two types most widely used are *distortion product otoacoustic emissions* (DPOAEs) and *transient evoked otoacoustic emissions* (TEOAEs). DPOAEs are the ones used in this thesis and they are further explained in section 2.3.3.

TEOAEs are evoked by a click stimulus which excites a broad region of the basilar membrane. Depending on the measurement device several types of stimuli are used, such as broad-band clicks, tone pips or tone bursts among others. The measurement procedure typically consists of repetitions of the preset stimulus. Then, the delayed cochlear responses in the ear canal are acquired and accumulated in a memory bank in order to enhance the detection of the small cochlear signals against the background noise.

TEOAEs are considered the most complex type of emission, and recent experiments suggest that TEOAE response might contain not only TEOAE components, but also distortion products and spontaneous emissions (Kalluri and Shera, 2001). TEOAEs are not normally seen in people with a hearing loss greater than 30 dB HL.

Currently, TEOAEs are clinically applied for the early detection of hearing loss in newborn and infants. Furthermore, there is a great international interest in their incorporation as part of a universal neonatal screening-program (Joint Committee on Infant Hearing, 2007).

### 2.3.3 Distortion product otoacoustic emissions

DPOAEs are a particular type of evoked otoacoustic emissions. For DPOAE measurements the sound probe delivers simultaneously two pure-tone stimuli called primaries  $f_1$  and  $f_2$ , with intensity levels  $L_1$  and  $L_2$  respectively. The nonlinear interaction of the primaries in the cochlea will evoke a series of combination tones, the most prominent normally observed at  $2f_1 - f_2$ . Therefore, DPOAE measurements consist typically on readings of the distortion product (DP) at  $2f_1 - f_2$ .

The level of the DP depends not only on the physiological conditions of the inner ear but also on the level of the primaries  $L_1$  and  $L_2$ , and the frequency ratio  $f_2/f_1$ . The strongest emissions are detected when using a frequency ratio  $f_2/f_1$  between 1.1 to 1.3. In humans, it is considered that the optimal ratio might be approximately  $f_1/f_2 = 1.22$ . This ratio is considered to provide the largest DPOAE levels for this separation of the two primary tones (Probst *et al.*, 1991).

The choice of the primary levels  $L_1$  and  $L_2$  might be a critical factor to assess cochlear function, and in most cases it depends on the purpose of the measurement. Two DPOAE protocols are typically employed. Protocols using primaries with equal intensities ( $L_1 = L_2$ ), and protocols using unequal intensities ( $L_1 > L_2$ ). The latter can better identify cases with hearing impairment and they are explained in section 2.3.4. Further explanation about the choice of  $L_1$  and  $L_2$ , and  $f_2/f_1$  in the experiments performed in this thesis is given in section 3.3.

For measurements of DPOAE over a wide frequency range along the basilar membrane, the primaries are typically swept in frequency. It is common to measure DPOAEs as low as about  $2f_1 - f_2 = 300$  Hz, although both acoustic noise from the environment and physiologic noise from the subject make DPOAEs less than 1 kHz difficult to measure. At high frequencies, DPOAEs can provide useful information to above  $2f_1 - f_2 = 6 - 8$  kHz, and most available equipment allows to perform measure-

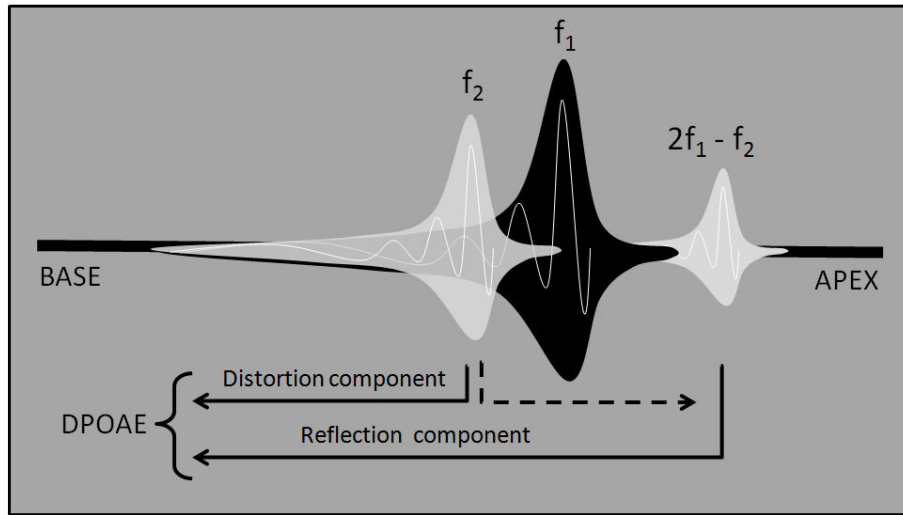


Figure 2.3: Conceptual diagram of the generation of the DPOAE fine structures according to the two-source model.

ments up to this limit, which is constrained primarily by the technical limitations of the loudspeakers (Robinette and Glatcke, 2000).

### Fine structures of the DPOAE

When DPOAE measurements are performed with sufficiently high frequency resolution, the  $2f_1 - f_2$  acoustic distortion product can show a particular pattern characterized by a series of amplitude peaks and valleys across frequency with peak-to-valley amplitude ratios as great as 20 dB (Gaskill and Brown, 1990; He and Schmiedt, 1993; Heitmann *et al.*, 1996; Reuter and Hammershøi, 2006) and a periodicity of  $3/32$  octaves (He and Schmiedt, 1993). This distinct characteristic in DPOAE levels with small frequency intervals is known as *fine structures*. Similar fine structures have been observed in hearing thresholds. However, it is not clear whether there is a unequivocal relation between the two (Talmadge *et al.*, 1998).

The reason for the fine structures is not completely understood but it is widely accepted that they reflect the interaction of two components: (1) the distortion component generated in the overlap region of the two-stimulus tones – which approximates to the region of  $f_2$ ; and (2) the reflection component generated at the  $2f_1 - f_2$  place. The DPOAE measured in the ear canal is assumed to be the vector sum of contributions arising from the  $f_2$  and  $2f_1 - f_2$  region (see Figure 2.3). The two components interfere with each other constructively or destructively, and as consequence, large level differences for small frequency intervals can be shown in the DPOAE. This hypothesis is known as the *two-source model* (Shaffer *et al.*, 2003; Mauermann *et al.*, 1999,b).

The distortion and reflection component may arise by two fundamentally different mechanisms within the cochlea: (1) nonlinear distortion; and (2) linear coherent reflection (for a detail description see Shera and Guinan, 1999). Nonlinear distortion arises from the action of the cochlear amplifier producing a wave-related mechanical interaction on the basilar membrane and depends on inherent physiological nonlinearities of the cochlear amplifier. Because this mechanism is associated with the traveling wave, it has historically been called a *wave-fixed* phenomenon (Kemp, 1986). In contrast, the reflection mechanism, often denoted as a *place-fixed* phenomenon, involves reflection of energy from *inhomogeneities* that are distributed

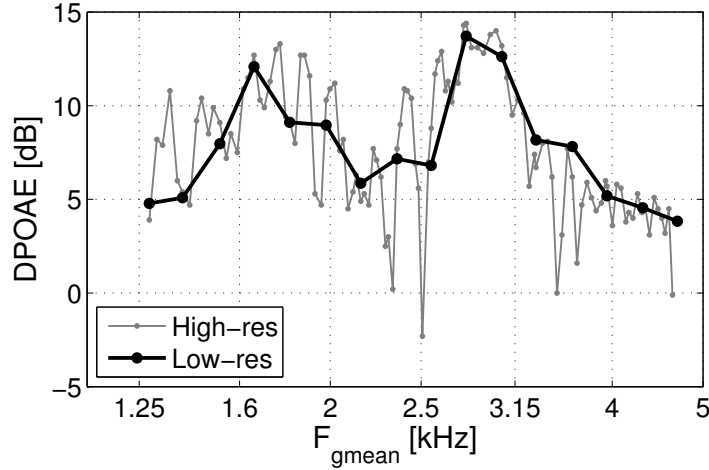


Figure 2.4: Example of a DPOAE measurement performed with low (Low-res) and high frequency-resolution (High-res). The fine structures are revealed for the High-res measurement.

randomly, but fixed in position, along the cochlear partition (Zweig and Shera, 1995). Although the nature of these inhomogeneities is not known, they are conceptualized as impedance irregularities or spatial corrugations in the cochlear mechanics or anatomy. Variation in the number and spacing of outer hair cells in primates has been suggested as an example of a micromechanical irregularity that could lead to reflection of energy. Such irregularities create reflections from multiple sites that sum with different phases. Only those reflections that sum constructively and arise from the tip of the basilar membrane excitation pattern in an active cochlea will have sufficient amplitude to be recorded in the ear canal as an emission (Zweig and Shera, 1995).

Figure 2.4 shows an example of a DPOAE measurement performed both with low and high frequency-resolution. The DPOAE data is plotted as a function of the geometric mean of the primaries ( $F_{gmean} = \sqrt{f_1 \cdot f_2}$ ). The fine structures are revealed for the measurement with high frequency-resolution. DPOAE measurements with low frequency-resolution can yield misleading interpretations because (1) they can fail to reveal changes in the properties of the fine structures that might be a sign of incipient hearing loss; and (2) the experimenter does not know whether a DP point -either with low or high-level- might fall into a minimum or maximum of the fine structures.

Measurement of the DPOAE fine structures are relatively time consuming. So far they have mainly been used for research purposes, usually selecting young subjects with normal hearing. For subjects of different age, the fine structure might be observable whenever the acoustic distortion-product is measurable, and its sharpness, defined as the peak-to-peak frequency distance, seems to be independent of age and hearing loss (He and Schmiedt, 1996).

Some authors suggest that the presence of fine structures is a property of the healthy ear and therefore their evaluation may reveal more information about the state of hearing than the DPOAE level alone – e.g., the fine structures reappear after a sudden hearing loss (Mauermann *et al.*, 1999b); and are reduced after aspirin consumption (Rao *et al.*, 1996). Other authors claim that the high variability usually seen in the pattern of the fine structures complicates the interpretation and

comparison of results across subjects (Shaffer, 2008; Heitmann *et al.*, 1996). Particularly critical is that the pattern of the fine structures get shifted along the frequency axis when the primary levels  $L_1$  and  $L_2$  are varied (He and Schmiedt, 1997) or the frequency ratio  $f_2/f_1$  is changed (Mauermann *et al.*, 1999; He and Schmiedt, 1997). This stimulus-dependent frequency shift complicates the comparison of DPOAE growth functions with audiometric thresholds and it might be responsible for the lack of correlation between DPOAEs and hearing thresholds reported by many researchers (Shaffer *et al.*, 2003; Heitmann *et al.*, 1996). For this reason, these authors are favorable to exclude the fine structures from the assessment.

At present, it is not clear whether the evaluation of the fine structures of the DPOAE might be critical for the detection of incipient hearing loss. Most of the experiments studying vulnerability of DPOAEs to noise exposures are performed with low frequency-resolution, insufficient to detect the fine structures of the DPOAE. Conclusions from these studies might have overlooked some important properties of the cochlea which are a sign of incipient hearing damage.

Two studies have investigated whether there are systematic changes in the DPOAE fine structures of humans after acoustic overexposure (Engdahl and Kemp, 1996; Reuter *et al.*, 2007). Both used a monaural narrow-band sound-exposure and monitored the fine structures at specific times during the recovery. Their results show that the most affected frequency range was located 1/2-octave above the exposure –similar to the *1/2-octave shift* seen in TTS experiments. However, they reported different effects in the fine structures. Engdahl and Kemp (1996) found for their two subjects that the maximum to minimum ratio of the fine structure decreased, and the whole pattern shifted toward lower frequencies after the exposure. Reuter *et al.* (2007) found for their 16 subjects that the effects were highly individual and no systematic change was observed –i.e., some subjects showed an increase in the depth of the fine structures while others showed a decrease, with no systematic shift in frequency.

#### 2.3.4 Influence of the primary levels $L_1/L_2$ in the assessment of NIHL

Several researchers have investigated the influence of the primary levels ( $L_1/L_2$ ) in order to find the most sensitive set of values to reveal changes in the characteristics of DPOAEs after noise overexposure. The current conception is that DPOAE levels show the greatest amplitude reduction when using low stimulus levels, with  $L_1 > L_2$  (Sutton *et al.*, 1994; Engdahl and Kemp, 1996; Gaskill and Brown, 1990; Hauser and Probst, 1991; Whitehead *et al.*, 1995; Marshall *et al.*, 2001). This effect is attributed to the highest functional activity of the OHCs for low-level stimulating tones.

Sutton *et al.* (1994) examined the effects of primary levels and ratio ( $L_1/L_2 = 60/60, 55/55, 60/30$ , and  $55/30$  dB SPL) for DPOAEs following a three-minute tonal monaural exposure to a 105 dB SPL tone at 2.8 kHz in 14 subjects. They reported that the combination  $L_1/L_2 = 55/30$  dB SPL was the most sensitive to overexposure effects. Four subjects received the same noise exposure a second time with hearing thresholds measured instead of DPOAEs. The time course of the recovery for DPOAEs ( $L_1/L_2 = 55/30$  dB SPL) and TTS were very similar. That is to say, subjects whose TTS was greatest displayed the greatest DPOAE loss; whereas for subjects whose TTS was unaffected there was no apparent DPOAE shift. These similarities suggest that with the appropriate parameters DPOAEs can be as sensitive to TTS as pure-tone audiometry.

In a similar experiment, Engdahl and Kemp (1996) exposed two subjects to a 1/3-octave narrow-band noise centered at 2 kHz during 10 minutes, and measured input/output (I/O) functions with  $L_2$  ranging from 50 to 75 dB SPL in steps of 5 dB.  $L_1$  was always 10 dB higher than  $L_2$ . DPOAEs were measured with  $f_2$  fixed near 3 kHz (1/2-octave above the noise exposure) and a frequency ratio  $f_2/f_1$  of 1.22. In agreement with Sutton *et al.* (1994), they found that the DPOAE amplitude reduction was greatest at low stimulus levels. They also noted that the DPOAE recovery functions were similar to TTS recovery functions in other studies.

Further, DPOAEs have been used as a screening tool to differentiate normal-hearing from impaired ears. Also in this case, researchers have reported a higher success when using primary levels with  $L_1 > L_2$  (Marshall *et al.*, 2001). Stover *et al.* (1996) reported that optimal  $L_2$  levels for separating normal-hearing from hearing-impaired ears were in the 50-60 dB SPL range, with  $L_1 - L_2 = 10$  dB.

### 2.3.5 Effect of impulsive noise on DPOAEs

Several animal studies have investigated the effects of industrial impulsive noise on DPOAEs. Results from animal studies cannot be extrapolated directly to humans. However, animal studies have remarkable advantages over human studies as they allow not only to use more severe exposure conditions but also to study morphological<sup>3</sup> and histopathological<sup>4</sup> changes in the inner ear.

Emmerich *et al.* (2000b) exposed six groups of guinea pigs to six different realistic-type industrial noises ranging from continuous to impulsive. Continuous noise stimuli consisted of broad-band noise, low and mid-frequency noise. All stimuli had the same energy. DPOAEs ( $L_1 = L_2 = 70$  dB SPL) were measured in a broad-band frequency range before and after the noise exposures. Their results showed that for the case of impulsive noise, DPOAE levels were significantly reduced in a broad-band frequency range, which were in good agreement with OHC loss. However, for the case of continuous noise, DPOAE levels were diminished according to the spectral content of the stimuli but with lesser coincidence of OHC loss. They concluded that different types of industrial noise tend to produce typical changes on DPOAE levels, being the impulsive type the most hazardous.

In addition, animal studies have shown that exposures to industrial impulsive noise can strongly damage OHCs, while the IHCs may remain intact (Emmerich *et al.*, 2000b,a). It has also been reported that for PTS below 25 dB, often there is no correlation between OHC loss and decrease of DPOAE level. However, for more severe permanent changes (PTS > 25 dB) there is a good correspondence between the two (Hamernik and Qiu, 2000). These observations suggest that more studies are necessary in order to understand how changes observed on DPOAEs (and OAEs in general) may relate to permanent hearing damage.

For humans, the literature regarding the effect of impulsive sounds on OAEs (either DPOAE or TEOAE) is still sparse and it has mostly focused on military settings (Marshall *et al.*, 2001; Attias *et al.*, 2001; Engdahl *et al.*, 1996; Konopka *et al.*, 2005; Miller *et al.*, 2004). Marshall *et al.* (2001) measured audiometric thresholds, DPOAEs ( $L_1/L_2 = 59/50$  and  $65/45$  dB SPL) and TEOAEs (74 dB pSPL click) in 285 U.S. Marine Corps recruits before and three weeks after exposure to impulse-noise military weapons. Peak levels ranged from 149 to 157 dB peak SPL. Audio-

<sup>3</sup>*Morphological*: Referring to the form and structure of organisms.

<sup>4</sup>*Histopathology*: The study of the microscopic anatomical changes in diseased tissue of an organism.



metric thresholds were measured at 0.5, 1, 2, 3, 4 and 6 kHz. OAEs were monitored in a similar frequency-range with low frequency-resolution. The results show that OAE levels (DPOAE and TEOAE) were significantly reduced after noise exposure, whereas no significant change was found for hearing thresholds. The authors concluded that OAEs were a more sensitive indicator of incipient hearing loss than audiometric thresholds. They also reported that for individual ears, subjects with low-level or absent OAEs before exposure were more likely to show a significant threshold shift after exposure to impulsive sounds. Similar findings were reported by Attias *et al.* (2001). However, in this case the noise exposures were not documented, which makes very difficult to conclude which characteristics of the impulsive exposures led to an increased NIHL.

An observation to the studies above is that perhaps longitudinal studies tend to show that OAEs have greater sensitivity to detect NIHL than hearing thresholds because pure-tone audiometries are performed with standard procedures. In these procedures, hearing thresholds may be determined in steps of 5 dB HL –excessive to detect immediate changes. Therefore, it can be argued that OAEs may be more sensitive than hearing thresholds to detect noise-induced damage when measured in the field, but it cannot be concluded that OAEs are more sensitive than hearing thresholds per se. For instance, laboratory studies have shown a more direct relationship between temporary changes in OAEs and hearing thresholds (TTS), which in part may be due to using higher-resolution Bekesy audiometry (Marshall *et al.*, 2001). Similarities have also been observed in the frequency specificity and time course of the recovery of TTS (e.g., Marshall *et al.*, 2001; Engdahl and Kemp, 1996; Emmerich *et al.*, 2000b; Reuter *et al.*, 2007). These similarities suggest that the underlying mechanisms behind changes in OAEs and hearing levels are closely related.

### 2.3.6 Choice of DPOAE over TEOAE

Both DPOAE and TEOAE have been successfully used to assess risk of NIHL, although literature is more extensive for DPOAE studies (for a review see Marshall *et al.*, 2001). However, at present there are not enough data to conclude which one of them is superior in detecting incipient damage in the inner ear.

The comparison between DPOAEs and TEOAEs in the assessment of risk of hearing loss is not straightforward as the results may depend on the set of parameters chosen for the measurements ( $L_1/L_2$  for DPOAEs and peak level pSPL of the click stimulus for TEOAEs). It might be for this reason that some authors have reported that TEOAE might be a better indicator of NIHL (Reshef *et al.*, 1993; Attias *et al.*, 1995; Prasher and Sulkowski, 1999); while other studies suggest that there is a certain range of parameters for which DPOAEs and TEOAEs perform similarly (Attias, 2000; Marshall *et al.*, 1998; Marshall and Heller, 1998).

Of special attention are the studies by Marshall *et al.* in which they compared the performance of DPOAE and TEOAE with TTS following a monaural tonal exposure of 2 kHz (105 dB SPL half-octave narrow-band noise at 1.414 kHz center frequency) in two different experiments with 14 human subjects. In the first experiment the authors measured TTS and DPOAE amplitude shifts interleaved along the same recovery function (Marshall *et al.*, 1998). DPOAE stimulus levels were  $L_1/L_2 = 70/60$  dB SPL and  $L_1/L_2 = 65/45$  dB SPL. The authors found a strong relationship between TTS and DPOAE amplitude shifts, as well as in the temporal characteristics of the recovery, particularly for the DPOAE stimulus  $L_1/L_2 = 65/45$  dB SPL. They

repeated the same experiment but using TEOAE instead of DPOAE (Marshall and Heller, 1998). TEOAEs were measured at 74 dB pSPL. Eight subjects participated also in the previous experiment, which allowed a better comparison of DPOAEs and TEOAEs. The authors found a strong relationship between TTS and TEOAE also in this case. When comparing the results from the eight subjects who participated in both experiments the authors reported that the maximum shift of both DPOAE and TEOAE was half an octave above the center frequency of the tonal exposure. They concluded that the underlying mechanism for amplitude shifts in DPOAE and TEOAE is closely linked to the underlying mechanism for TTS.

Knight and Kemp (2000) indicated that the issue is not to choose between DPOAEs and TEOAEs, but rather to decide what underlying physiological mechanisms are being targeted and to attempt to choose the stimulus parameters accordingly (Knight and Kemp, 2000). In the present dissertation, one of the main objectives was to continue with the research performed at the Section of Acoustics of Aalborg University regarding the evaluation of NIHL with DPOAEs and improve our understanding of the properties and generation mechanisms of the fine structures of the DPOAE. It is for this reason that DPOAEs were chosen over TEOAEs.

# Chapter 3

## Methods

### 3.1 Methodological overview

Experiments were done at the Section of Acoustics<sup>1</sup> of Aalborg University (AAU<sup>2</sup>) and they were approved by the *Danish National Ethical Committee on Biomedical Research Ethics* (CVK<sup>3</sup>). All sessions were conducted in a double-walled, sound-isolated audiometry-chamber which complies with the background noise requirements stated in ISO 8253-1:1989. The experimental protocol in both experiments was similar. A total of 16 normal-hearing subjects participated in each experiment. The screening for normal hearing consisted of measurements of pure-tone audiometry, tympanometry and middle-ear reflex activity. Subjects also underwent an interview in order to obtain more information about their noise exposure history, previous hearing problems and reaction to noise, e.g., annoyance or headaches. Only subjects who reported neither known incidents of excessive noise exposures nor abnormal reactions to noise were allowed to participate. A pure-tone audiometry was performed at the end of each experimental session to verify that the hearing of the subjects recovered completely.

Subjects were exposed to a particular noise stimuli for 10 minutes via a pair of headphones model Sennheiser HDA 200. DPOAEs were measured before and after the noise exposure. DPOAEs were measured only in one ear -either left or right- which was balanced across subjects.

The main differences between the experiments consisted of:

- PAPER I: subjects were exposed to a monaural pure tone of 2 kHz ( $L_{EX,8h} = 80$  dBA). In the following, this stimulus is referred to as TONE. DPOAEs were measured during the following 70 minutes after the exposure. The protocol allowed to monitor the fine structures of the DPOAE in a 1/3-octave band centered at 3 kHz.
- PAPER II: subjects were exposed to two types of binaural industrial-recordings in two different sessions: (1) a continuous broad-band noise normalized to  $L_{EX,8h} = 80$  dBA (crest factor  $CF = 13$  and kurtosis  $\beta = 3$ ); and (2) the combination of the previous stimulus (scaled down in level) with an impulsive noise normalized to  $L_{EX,8h} = 75 + 5_{dBpenalty} = 80$  dBA ( $L_{peak} = 117$  dBC; repetition rate of 0.5 impacts per second,  $CF = 25$ ,  $\beta = 32$ ). Both noise

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<sup>1</sup><http://www.es.aau.dk/sections/acoustics/>

<sup>2</sup><http://en.aau.dk/>

<sup>3</sup>Den Centrale Videnskabetiske Komité – <http://www.cvk.sum.dk>



stimuli were bandpass filtered from 100 Hz to 16 kHz. In the following, the stimuli are referred to as CONT and IMP+CONT respectively. DPOAEs were measured in a broad frequency range ( $1.25 < F_{gmean}[\text{kHz}] < 5$ ) during the following 90 minutes of recovery after the exposure.

## 3.2 Pure-tone audiometry and middle-ear test

Pure-tone audiometries were measured using the commercial audiometer *Madsen Orbiter 922* and the audiometric headphones *Telephonic TDH39*. The left ear was tested first. Hearing thresholds were determined in a frequency range from 250 Hz to 8 kHz in octave intervals by using the ascending method, which complies with the norms for automatic audiometries ISO 8253-1:1989. Tympanometries and stapedi-reflex tests were performed with the *Interacoustics impedance audiometer AT235*.

## 3.3 DPOAE assessment

A new methodological protocol was programmed with the commercial system ILO96 from Otodynamics<sup>4</sup> by using the *DPOAE macro* operating mode. The protocol allows DPOAE measurements with a higher time and frequency resolution, and more programming flexibility than the one used in previous experiments at Aalborg University (Reuter and Hammershøi, 2006, 2007; Reuter *et al.*, 2007). This is particularly important in order to monitor sudden changes during the recovery process of DPOAEs after noise overexposure. The *DPOAE-macro mode* allows the user to program the operations to be performed by the system by writing a function called *macro*. The following parameters can be programmed: up to 16 user-defined pairs of primaries  $f_1$  and  $f_2$ ; level of the primaries  $L_1/L_2$ ; frequency ratio  $f_2/f_1$ ; averaging time per primaries presentation; and measurement time in loop-mode.

The main advantage of the DPOAE-macro mode for the assessment of DPOAE recovery is that the measurement process becomes highly automated, and data acquisition can be performed in a loop-mode without interruptions. However, it has the limitation that it only allows programming a maximum of 16 user-defined pairs of primaries  $f_1$  and  $f_2$ . Therefore, our measurements in both experiments are limited to this number of primaries.

Although chronologically the implementation of the *DPOAE-macro mode* was performed first, it is presented in PAPER III due to its minor relevance to understand PAPERS I and II.

Individual macros were programmed for each subject. The only difference between macros is the presentation order of the primaries, which was counter-balanced across subjects by means of a Latin-square design.

### Choice of primary levels and ratio

In both experiments, DPOAE measurements were performed using a ratio of  $f_2/f_1 = 1.22$ . This ratio was chosen as it may provide the largest DPOAE levels across subjects (Probst *et al.*, 1991). The primary levels were set to  $L_1/L_2 = 65/45$  dB. This choice is a compromise between:

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<sup>4</sup><http://www.otodynamics.com/>

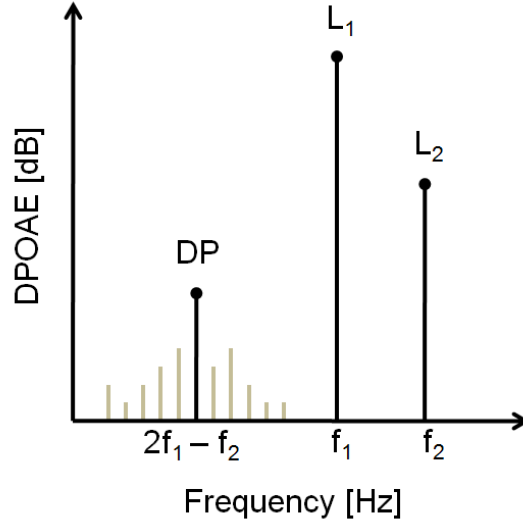


Figure 3.1: Schematic representation of the primaries  $f_1$  and  $f_2$  for the assessment of DPOAE. The DP frequency at  $2f_1 - f_2$  is also shown. The primaries are presented with a frequency ratio of  $f_2/f_1 = 1.22$  and fixed primary levels of  $L_1/L_2 = 65/45$  dB. The noise, estimated from the ten Fourier components nearest to but not including the  $2f_1 - f_2$  frequency, is shown as gray lines.

1. High sensitivity to detect small changes after noise overexposure (see section 2.3.4).
2. Measurable DPOAEs: Whitehead *et al.* (1995) measured DPOAEs for various primary-level combinations at different frequencies and reported that 65/45 dB showed relatively high level DPOAEs for all tested frequencies;
3. Detect the presence of fine structures: measurements with varying equal primary levels ( $L_1 = L_2 = 40$  to 70 dB) have shown a flattening of the fine structure for some subjects at high levels (He and Schmiedt, 1993; Heitmann *et al.*, 1996).

The averaging time per each pair of primaries presented was set to 1.3 seconds. With this configuration the ILO96 takes 28 seconds to measure the 16 pairs of primaries in each loop. Prior to each measurement the ILO96 verifies the correct fitting of the sound probe in the ear canal by a checkfit procedure. During the checkfit two broadband-click stimuli are alternately delivered by the two output transducers of the sound probe. The checkfit result is stored in an array and used during data collection to balance and normalize the two stimuli levels. All spectrum analyses are done by the ILO96, which performs a fast Fourier transform FFT with a frequency resolution of 12.2 Hz. The noise is estimated from the ten Fourier components nearest to but not including the  $2f_1 - f_2$  frequency. The noise is represented as all levels within two standard deviations of the background noise, i.e., the limits of the 95% confidence region. An schematic illustration of the primaries  $f_1$  and  $f_2$ , the distortion product  $2f_1 - f_2$  and the estimated noise floor is shown in figure 3.1.

### Choice of DPOAE frequency-range assessment

The main difference for the assessment of DPOAEs in the experiments in PAPER I and II is the frequency range of the measurement and the spacing of the primaries.

$f_1$	$f_2$	$2f_1 - f_2$	$F_{gmean}$	$f_2/f_1$
2441	2979	1903	2697	1,22
2478	3027	1929	2739	1,22
2527	3076	1978	2788	1,21
2563	3125	2001	2830	1,21
2600	3174	2026	2873	1,22
2637	3223	2051	2915	1,22
2686	3271	2101	2964	1,21
2722	3320	2124	3006	1,22
2759	3369	2149	3049	1,22
2808	3430	2186	3103	1,22
2856	3479	2233	3152	1,21
2893	3528	2258	3195	1,21
2930	3577	2283	3237	1,22
2966	3625	2307	3279	1,22
3015	3674	2356	3328	1,21
3052	3723	2381	3371	1,22

Table 3.1: Frequency value [Hz] of the 16 pairs of primaries  $f_1$  and  $f_2$  used in the first experiment (PAPER I). Their distortion product  $2f_1 - f_2$ , geometric mean  $F_{gmean}$ , and frequency ratio  $f_2/f_1$  are also shown. For some primaries the frequency ratio is 1.21 instead of the programmed value of 1.22. This is due to limitations of the measuring system ILO96 – i.e., the ILO96 presents primaries at discrete frequencies with a minimum resolution of 12 Hz. Thus, when the system presents  $f_1$  it selects the value of  $f_2$  that will yield the closest value to 1.22.

In the first experiment (PAPER I), DPOAE measurements are performed in a narrow-band frequency range with high frequency-resolution. It was expected that a tonal exposure would produce the highest impact on the basilar membrane 1/2-octave above the exposure frequency. This phenomenon, known as the *half-octave shift*, was first observed by Davis *et al.* (1950) when measuring TTS on human subjects and they reported that the predominant hearing loss resulting from exposure to sufficiently intense pure tones is one-half to one octave above the stimulating frequency. Several researchers have confirmed the half-octave shift in experiments assessing TTS (Ordoñez, 2005) and PTS after tonal or narrow-band exposures (Mills *et al.*, 1979). The half-octave shift has also been observed for measurements of DPOAE and TEOAE (Engdahl and Kemp, 1996; Reuter *et al.*, 2007; Marshall *et al.*, 2001). However, in this case the authors have reported that OAE levels may be reduced in a broader frequency range than the one typically seen in TTS measurements. The authors attribute this difference to the higher sensitivity of OAEs to detect early hearing loss.

The stimulus used in PAPER I consisted of a pure tone of 2 kHz. Hence, DPOAEs were measured in a frequency range centered at 3 kHz ( $F_{gmean}$ ) –i.e., half-octave above the exposure tone. As the purpose of the experiment was to monitor the fine structures of the DPOAE, the frequency range was covered by 16 equally-spaced pairs of primaries with a frequency resolution of 48 Hz. With this configuration the frequency range covered approximately a 1/3-octave band centered at 3 kHz. Table 3.1 shows the frequency value of the 16 pairs of primaries.

A tonal exposure was chosen in an effort to minimize the region of the basilar membrane affected after overexposure. Further, it was decided to use a pure tone of 2 kHz based on the results of a pilot test, in which a pure tone of 1 kHz was used instead. Two normal-hearing subjects participated in the pilot test. One of their ears was exposed to a pure tone of 1 kHz during 10 minutes. The exposure level was normalized to  $L_{EX,8h} = 80$  dBA, which corresponds to an  $L_{ear,10min}$  of 96.2 dBA measured at the blocked-entrance ear canal according to ISO 11904-1:2002. DPOAEs were measured before and during the following 20 minutes after the exposure in a frequency range centered at 1.5 kHz (half-octave above the exposure frequency) with the DPOAE-macro mode. The results showed that DPOAEs from both subjects had a poor S/N in the measured frequency range. The low S/N was due to low DPOAE levels –which may be due to the particular physiological hearing condition of the subjects– and also a high background noise during the measurements. A low S/N may be detrimental for the analysis of the results. This is particularly critical for the minima of the fine structures, as they might appear below the background noise and therefore, influence their analysis. Therefore, it was decided to use a pure tone of 2 kHz instead in an attempt to increase the S/N ratio during DPOAE measurements. This decision is based on the following considerations:

1. The high background noise in DPOAE measurements is typically seen at low frequencies. This noise is attributed not only to acoustic noise from the environment but also to physiological noise from the subject due to breathing, swallowing and/or heartbeats among others. Thus, it was expected that DPOAE measurements in a higher frequency range will be less affected by background noise.
2. Results from Reuter and Hammershøi (2006) corresponding to the average of 50 normal-hearing subjects show that DPOAE levels are lower in the low frequency-range. Thus, it was expected that subjects will show higher DPOAE levels at higher frequencies.

Only one of the subjects in the pilot test showed an evident DPOAE decrement after exposure. It was also observed that for this subject DPOAEs recovered gradually after the exposure. However, DPOAEs did not recover completely by the end of the observation period of 20 minutes, as they were still approximately 2 dB below pre-exposure levels. For this reason, it was decided to increase the observation period in the formal experiment to a duration of 70 minutes in order to allow complete restitution of DPOAE levels. For this subject, DPOAEs were measured again the next day and they were fully recovered. Finally, none of the subjects in the pilot test had measurable fine structures.

In the second experiment (PAPER II) it was expected that the broadband noise stimuli CONT and IMP+CONT would have an impact on a wide region of the basilar membrane. Hence, DPOAE measurements were performed in the frequency range  $1282 < F_{gmean}[\text{Hz}] < 4695$  with a frequency resolution of eight points per octave. This frequency range is a compromise between measuring DPOAEs in a broad region while avoiding low S/N at lower frequencies, and a poor probe fitting at high frequencies. Nevertheless, it was anticipated that subjects may show lower S/N at lower frequencies for the reasons explained in the previous section.

Table 3.2 shows the frequency value of the 16 pairs of primaries.

$f_1$	$f_2$	$F_{gmean}$	$2f_1 - f_2$	$f_2/f_1$
1160	1416	1282	904	1,22
1270	1538	1398	1002	1,21
1379	1685	1524	1073	1,22
1501	1831	1658	1171	1,22
1636	2002	1810	1270	1,22
1794	2185	1980	1403	1,22
1953	2380	2156	1526	1,22
2124	2600	2350	1648	1,22
2319	2832	2563	1806	1,22
2527	3088	2793	1966	1,22
2759	3369	3049	2149	1,22
3003	3662	3316	2344	1,22
3284	4004	3626	2564	1,22
3577	4358	3948	2796	1,22
3894	4761	4306	3027	1,22
4248	5188	4695	3308	1,22

Table 3.2: Frequency value [Hz] of the 16 pairs of primaries  $f_1$  and  $f_2$  used in the second experiment (PAPER II). Their distortion product  $2f_1 - f_2$ , geometric mean  $F_{gmean}$ , and frequency ratio  $f_2/f_1$  are also shown. For some primaries the frequency ratio is 1.21 instead of the programmed value of 1.22. This is due to limitations of the measuring system ILO96 – i.e., the ILO96 presents primaries at discrete frequencies with a minimum resolution of 12 Hz. Thus, when the system presents  $f_1$  it selects the value of  $f_2$  that will yield the closest value to 1.22.

## 3.4 Selection of noise stimuli

Some of the characteristics of the stimuli TONE, CONT and IMP+CONT were kept identical in order to facilitate the comparison between them. These characteristics are (1) the duration of the exposure; and (2) the exposure level  $L_{EX,8h}$ . Their choice is explained next.

### 3.4.1 Choice of exposure level

The choice of the exposure level ( $L_{EX,8h} = 80$  dBA) for the stimuli TONE and CONT was based on the fact that it is ethically unacceptable to induce a permanent damage in the hearing of the subjects during the noise exposures. According to ISO 1999:1990 a daily eight-hour exposure during 10 years to 80 dBA will not result in a significant hearing loss in any percentage of the population. However, this conclusion is based on longitudinal studies in which workers were exposed mainly to continuous broad-band noise, and it is not certain that it will also be safe for all types of impulsive exposures. For this reason, instead of normalizing the stimulus IMP+CONT to 80 dBA (this is, the same  $L_{EX,8h}$  as the stimuli CONT and TONE) and study whether the effects of the three equal-energy stimuli could be compared, it was preferred to use a more conservative approach and normalize the stimulus IMP+CONT to 75 dBA. In this way, the stimulus IMP+CONT can still be penalized +5 dB for impulsiveness and thereby, its corrected exposure level becomes  $L_{EX,8h} = 75 + 5_{dBpenalty} = 80$  dBA.

For the stimulus TONE, an exposure level of  $L_{EX,8h} = 80$  dBA corresponds to an equivalent continuous A-weighted sound-pressure level (SPL) of  $L_{Aeq,10min} = 96.8$  dB according to ISO 1999:1990. This level corresponds to a  $L_{ear}$  of 102.2 dB measured at the blocked-entrance ear canal according to ISO 11904-1:2002.

For the stimuli CONT and IMP+CONT further information is given in section 3.5.

### 3.4.2 Choice of stimuli duration

Noise stimuli had a duration of 10 minutes. This duration is a compromise between avoiding too short or too long exposures and still being able to induce a temporary change on the hearing of the subjects.

Noise exposures shorter than 10 minutes may introduce the following disadvantages:

1. According to literature, intense tonal and narrow-band exposures of approximately less than four minutes will produce a peak in the TTS recovery pattern at approximately two minutes after the end of the noise exposure (Hirsh, 1958; Quaranta *et al.*, 1998; Botsford, 1971). This peak is known as the *two-minute bounce* and it seems to disappear for longer exposures (Patuzzi, 1998; Botsford, 1971). A similar phenomenon has also been reported in the DPOAE recovery data from animal studies (Kirk *et al.*, 1997; Kirk and Patuzzi, 1997). Some authors have suggested that the *two-minute bounce* might be the product of the interaction between different short-lived recovery mechanisms that may reflect different metabolic or mechanical changes in the cochlea. The *two-minute bounce* may complicate the comparison of the results between tonal and broad-band noise exposures.



2. Noise exposures would require higher SPL if an exposure level ( $L_{EX,8h}$ ) of 80 dBA is to be maintained. This could be particularly critical in the case of the stimulus IMP+CONT, as peak levels would be higher than 120 dBC and the risk of inducing a permanent hearing loss would increase.

Noise exposures longer than 10 minutes are avoided for the following reasons:

- To reduce the total number of impulses within the exposure: Impulses from the stimulus IMP+CONT had a repetition rate of 0.5 impacts per second. Such a repetition rate may pose a high fatigue on the auditory system (see section 2.1.4). For this reason, it was decided to avoid long-duration stimuli in order to lower the total number of stimuli within an exposure.
- To avoid long experimental sessions that might be exhausting for subjects. Tiredness and fatigue may result in DPOAE measurements of poor quality, as subjects may cause involuntary body movements and/or deep inhalation.

### 3.4.3 Noise stimuli CONT and IMP+CONT

The noise stimuli CONT and IMP+CONT consisted of binaural recordings of industrial noises with the following properties:

#### Continuous noise (CONT)

The stimulus CONT was an industrial noise of continuous character. The stimulus consisted of the mix of a bench-driller and a table-saw. Both machines radiate noise of continuous character but in different frequency range; i.e., low frequency for the driller and mid-high frequency for the saw. It was decided to mix the two noise sources in order to cover a broader frequency range during the noise exposure. Noise recordings were done separately and mixed afterwards via software. The 10 minute-duration stimulus was obtained by concatenating a segment of 30 seconds from the original recordings. A crossfading technique was applied in order to have a smooth and unnoticeable transition between segments. The exposure level was normalized to an eight-hour working day  $L_{EX,8h}$  of 80 dBA (ISO 1999:1990). This level corresponds to  $L_{ear,10min} = 102.7$  dB measured at the blocked-entrance ear canal (ISO 11904-1:2002).

#### Impulsive + continuous noise (IMP+CONT)

The stimulus IMP+CONT consisted of the combination of impulsive and continuous industrial noise. The continuous noise was a scaled version in level of the aforementioned stimulus CONT. The impulsive noise was a hammer beating a metal plate with a repetition rate of 0.5 impacts per second. Thus, there are a total of 300 impulses within the exposure. The 10-minute-duration sequence of impulses consisted of duplicates of one single hammer impact selected from the original recordings. In this manner, it is assured that the impulsive parameters remained constant throughout the exposure. The impact chosen had a peak level of 117 dBC; rise time of 1.64 msec; and a B-duration of 70 msec. After the addition of the continuous noise CONT, peak levels had a standard deviation of 0.3 dBC. The exposure level ( $L_{EX,8h}$ ) was normalized to 75 dBA (ISO 1999:1990). This level corresponds to  $L_{ear,10min} = 98.2$  dB measured at the blocked-entrance ear canal (ISO 11904-1:2002).



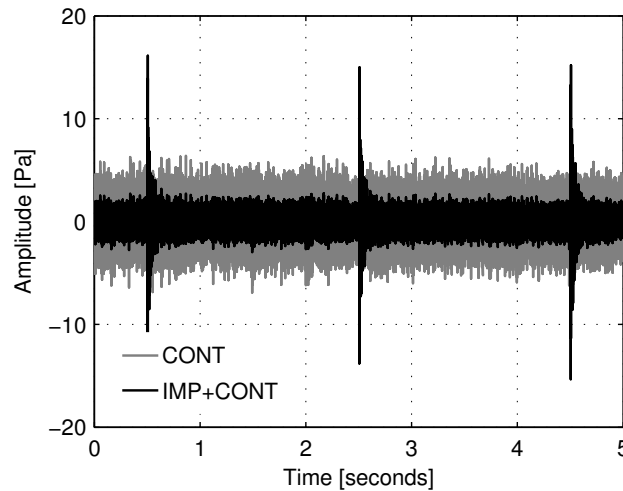


Figure 3.2: Five-second excerpt of the diffuse-field waveforms of stimuli CONT and IMP+CONT.

According to ISO 1999:1990 and the guidelines provided by the *Danish Working Environment Authority*, the stimulus IMP+CONT can be penalized for impulsiveness. Therefore, the corrected exposure level is  $L_{EX,8h} = 75 + 5_{dB_{penalty}} = 80$  dBA.

The combination of impulsive and continuous noise was preferred over a purely impulsive stimulus (IMP) in order to have similar spectral energy in both stimuli CONT and IMP+CONT. The individual stimuli IMP and CONT have different noise spectra. Most of the energy content of the stimulus IMP is concentrated around 4 kHz, whereas the stimulus CONT covers a broad-band frequency range. By adding the two stimuli together, both stimuli CONT and IMP+CONT have a similar spectral content.

Figure 3.2 shows the diffuse-field related waveforms of the stimuli CONT and IMP+CONT. Figure 3.3 shows on top the diffuse-field-related value of both stimuli in 1/3-octave-bands; and at the bottom the relative difference between them normalized at 1 kHz. It can be seen that both stimuli have similar noise spectra except around 4 kHz due to the higher energy contribution of the hammer impact in that range.

#### 3.4.4 Binaural recordings, sound processing and reproduction

Binaural technology is chosen for the recording and reproduction of real-life noise stimuli in order to reproduce realistic noise exposures in laboratory conditions. That is, to expose subjects to the original exposure by preserving the spatial information during the recordings and reproduce the effect of proximity to the sound source. This is particularly important when considering the role of the acoustic middle-ear reflex during the noise exposures, and the fact that the incoming sound direction might have an effect on the bilateral interaction of the stapedius muscle; i.e., the contraction of the muscle in one ear is influenced by the state of the muscle in the other ear (Møller, 2006).

Binaural recordings were done in a mechanical workshop at Aalborg University with the artificial head *Valdemar* (Christensen and Møller, 2000; Minnaar *et al.*, 2001) connected to the multi-channel measuring system *Harmonie* by *01dB-*

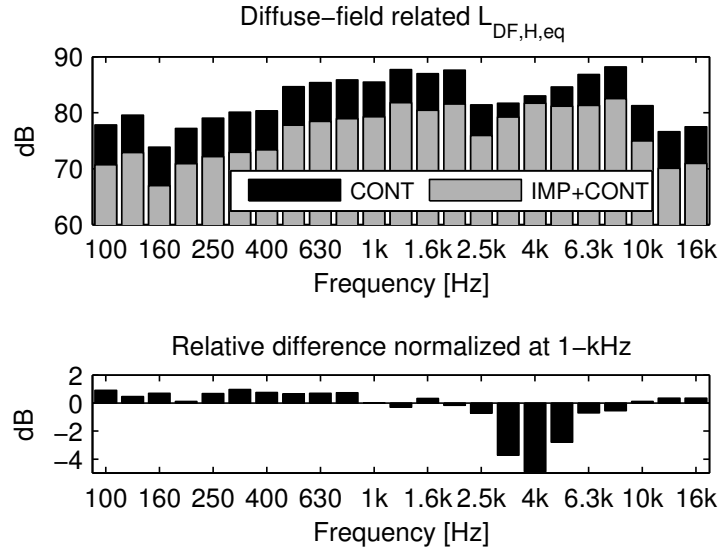


Figure 3.3: Top: diffuse-field related ( $L_{DF,H,eq}$ ) in 1/3-octave bands for stimuli CONT and IMP+CONT. Bottom: relative difference between the previous levels normalized at 1 kHz.

*Metravib.* The artificial head was placed at the position normally occupied by the operator of the machine, with the operator absent. For recordings of the hammer impacts the artificial head was placed in front of the operator; so that the operator and the artificial head were symmetrically located with respect to the impact. With this configuration the interaural time and level differences (ITD and ILD) between the left and right channel are minimal (Blauert, 1997). The recordings were done with a sampling frequency of 51.200 Hz and analog-to-digital (A/D) converters of 24-bit resolution.

Signal post-processing was done in Matlab. Each channel of the binaural recordings (L-R) was processed separately to obtain the desired parameters of the stimuli CONT and IMP+CONT. The ITDs between the left and right channel from the original recordings were kept. The original ILD differences computed over a period of 30 seconds were within  $\pm 1.5$  dB for both stimuli. This imbalance in the presentation level between left and right ears might bias the results of the experiment. Therefore, it was decided to eliminate the ILDs and have the same presentation level in both channels. This correction may induce a minor alteration in the localization of the virtual sound source (Blauert, 1997).

Noise stimuli were bandpass filtered from 100 Hz to 16 kHz. Diffuse-field related levels ( $L_{DF,H,eq}$ ) were derived from binaural recordings by means of a high-pass FIR filter which complies with the specifications given in the standard ISO 11904-1:2002. More information on the filter properties and procedure can be found in Ordoñez (2005, appx. E). For a correct binaural reproduction (Møller, 1992), the headphones were equalized from 100 Hz to 16 kHz according to the equalization filter described by Ordoñez (2005, appx. E).

### 3.4.5 Choice of impulsive characteristics

The criteria for choosing the impulsive characteristics of the stimulus IMP+CONT were:

- It is an imperative requirement that the stimulus is innocuous for the hearing of the subjects; yet it induces temporary changes in the characteristics of the DPOAEs.
- The stimulus must allow penalization of +5 dB for impulsiveness according to the guidelines provided by the *Danish Working Environment Authority* (Arbejdstilsynet, 2003) –i.e., peak levels may be higher than 115 dBC (or dBA) more than once per minute (Arbejdstilsynet, 2003).
- The crest factor and kurtosis values must be distinct from those of continuous signals, so that the stimulus IMP+CONT can be clearly considered of impulsive character.

Eight different noise stimuli were created. They ranged from continuous broadband to purely impulsive, through different combinations of impulsive+continuous noise. The stimuli consisted of 30 second segments<sup>5</sup> taken from the stimuli CONT and IMP described in section 3.4.3. In the following, the stimuli are labeled from S1 to S8. S1 was the the stimulus CONT. S8 was the stimulus IMP. The rest of the stimuli consisted of combinations of IMP+CONT.

The purpose was (1) to investigate how the parameters crest factor and kurtosis vary depending on the impulsiveness of the signal; and (2) to use the AHAAH model in order to obtain additional information regarding the risk of hearing loss of the impulsive stimuli on the hearing of the subjects.

According to the cumulative-crest-factor method proposed by Stark and Pekkari-nen (1987), the crest factor is calculated as  $L_{Apeak} - L_{Aeq}$ . Nevertheless, the authors reported that the A-weighting has only a slight effect in peak levels of different frequency content for most industrial environments. In our study, we used  $L_{Cpeak}$  instead for the calculation of the crest factor. This option was preferred as ISO 1999:1990 establishes C-weighted peak levels as the main descriptor of the impulsive characteristics of acoustic signals.

The peak level, crest factor and kurtosis of the eight noise stimuli are shown in table 3.3. Peak levels for the impulsive stimuli ranged from 114 to 119 dBC in steps of 1 dB. The stimuli S2–S8 were normalized to  $L_{EX,8h} = 75$  dBA. It can be seen that the crest factor increases in a linear manner from S1 to S8, whereas the kurtosis increases exponentially. It can also be seen that the kurtosis of the continuous noise (S1) is 3 –the same as a Gaussian noise– whereas the kurtosis of the purely impulsive signal (S8) is 213. Further, when the continuous noise is added with a crest factor of 27 (S7), the kurtosis is markedly lower at 97.6.

### AHAAH analysis

The risk of hearing loss of the stimuli S2–S8 was studied by using the AHAAH model. The purpose of the analysis was to select a peak level and number of impulses to be used in the formal test that will not result in a permanent damage in the hearing of the subjects. Despite the limitations of the AHAAH to predict risk of hearing loss from occupational exposures (see section 2.2.4), the model was used because it provides an insight into the mechanical impact that the noise stimuli may have

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<sup>5</sup>The 10 minute-duration stimulus CONT was obtained by concatenating a segment of 30 seconds from the original recordings. Therefore, 30-second segments were chosen for the analysis in order to include all the possible variations of the original waveform.

	Noise stimuli							
	S1	S2	S3	S4	S5	S6	S7	S8
$L_{Cpeak}$	110	114	115	116	117	118	119	120
CF	13	22	23	24	25	26	27	28
$\beta$	3	7.2	12.3	19.5	32	68.8	97.6	213

Table 3.3: C-weighted peak level ( $L_{Cpeak}$  [dB]), crest factor (CF [dB]) and kurtosis ( $\beta$ ) of the eight noise stimuli (S1–S8). S1 is a continuous broad-band noise (CONT). S8 is a purely impulsive noise (IMP). S2–S7 are combinations impulsive+continuous noise (IMP+CONT).

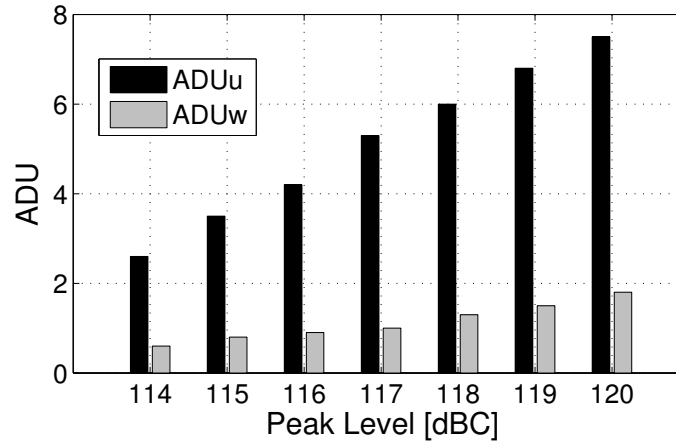


Figure 3.4: Number of ADUs produced by one single exposure to the stimuli S2–S8 for both warned (ADUw) and unwarned (ADUu) stapedius reflex. The stimuli are presented as a function of their peak level.

on the inner ear. In this way, it is possible to compare their hazardous effects and determine which stimuli might pose a higher risk of hearing loss.

Due to computational limitations, the AHAH software allows analysis of time-pressure signals with a maximum duration of approximately 800 milliseconds. Hence, segments of 800 milliseconds were selected from the stimuli S2:S8. Each segment contained the main energy of an impulse plus the contribution of the continuous noise in the tail, except for the case of the purely impulsive noise (S8).

The stimuli were analyzed with the AHAH software. For each stimulus the following parameters were obtained:

**ADUw (Auditory Damage Unit warned):** Auditory Damage Unit from one single impulse with activated stapedius reflex (warned).

**ADUu (Auditory Damage Unit unwarned):** Auditory Damage Unit from one single impulse with deactivated stapedius reflex (unwarned).

**Nw (Number warned):** Maximum number of exposures with activated stapedius reflex that are considered safe. In other words, number of necessary exposures based on the value of ADUw to reach 500 ADUs.

**Nu (Number unwarned):** Maximum number of exposures with deactivated stapedius

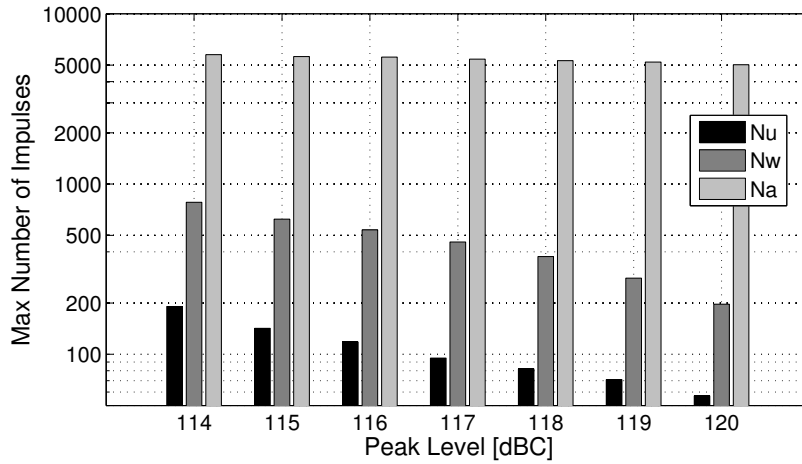


Figure 3.5: Maximum number of impulses of stimuli S2–S8 under the conditions warned (Nw) and unwarned (Nu) stapedius reflex; and according to the A-weighting principle (Na).

reflex that are considered safe. This is also the number of necessary exposures based on the value of ADUu to reach 500 ADUs.

**Na (Number A-weighted):** Maximum number of exposures according to the A-weighted energy. This number is calculated based on estimations of the acoustic energy in  $J/m^2$  (Joule per square meter). The AHAH considers that exposures above  $8.7 J/m^2$  are hazardous for the auditory system. Therefore, Na is the number of necessary exposures to reach this value.

Figure 3.4 shows the ADUs produced by one single exposure to each stimulus for both warned (ADUw) and unwarned (ADUu) stapedius reflex. The stimuli are presented as a function of the peak level so that it is easier to visualize the influence of the peak level in the number of ADUs. It can be seen that the higher the peak level, the higher the number of ADUs. It can also be seen that as the peak level increases, the growth of ADUs is higher for unwarned exposures.

Figure 3.5 shows the maximum number of impulses that a person can tolerate without introducing a permanent hearing damage for the stimuli S2–S8 of the analysis based on estimations of Nu, Nw and Na. Results are presented as a function of the peak level of the stimuli. The number of impulses is displayed in a logarithmic scale due to the high differences in the outcome. Thus, according to the A-weighting principle the six stimuli allow an exposure to approximately 5000 impulses. However, based on prediction of ADUs for warned and unwarned exposures the number of impulses goes from 50 to 700, depending on the stimulus.

The results of the analysis show that there are high discrepancies between the maximum number of tolerable impulses based on ADU estimations from the AHAH, and the A-weighted principle. Thus, the AHAH is more conservative and it recommends a significantly lower number of exposures than the A-weighting. These discrepancies may be due to the fact that the AHAH was not tuned to predict risk of hearing loss from low-level impulses that do not have a direct mechanical impact on the inner ear. The results also show that the peak level influences the maximum number of impulses that the auditory system can tolerate. The higher the peak level, the higher the mechanical impact on the basilar membrane and, therefore, the lower the number of impulses recommended.

### Conclusions from the AHAH analysis

The final stimulus IMP+CONT chosen for the experiment in PAPER II was the stimulus S5. This stimulus has a C-weighted peak level of 117 dB, a crest factor of 25 dB and a kurtosis  $\beta$  of 32. As the peak level is above 115 dBC, the stimulus can be penalized +5 dB for impulsiveness according to the guidelines provided by the *Danish Working Environment Authority* (Arbejdstilsynet, 2003).

A peak level of 117 dBC was chosen because it is a compromise between (1) avoiding a higher risk of hearing loss for higher peak levels; and (2) still being able to induce temporary changes in the properties of the inner ear. In addition, according to the results depicted in figure 3.5, a peak level of 117 dBC will allow a total of 5423 impulses based on A-weighted measurements, 457 impulses with a warned middle-ear (Nw), and 95 impulses with unwarned middle ear (Nu). For this reason, it was decided to limit the number of impulses in the stimulus IMP+CONT to a total of 300 impulses. This number is a compromise between the 457 and 95 impulses recommended from warned (Nw) and unwarned (Nu) middle-ear. As the stimuli used in the formal test were set to have a duration of 10 minutes, the 300 impulses were presented with a repetition rate of one impulse every two seconds.

A crest factor of 25 dB is higher than the fence value of 15 dB established by the cumulative crest factor method. As the 10-minute stimulus IMP+CONT has a constant crest factor of 25 dB, it can be considered impulsive throughout the exposure. In a similar manner, it is considered that a kurtosis of 32 is clearly distinct from that of the continuous-broad-band stimulus CONT ( $\beta = 3$ ).

#### 3.4.6 Binaural vs monaural noise exposures

Both stimuli CONT and IMP+CONT were presented binaurally, whereas the stimulus TONE was presented monaurally. The difference in the presentation method (binaural/monaural) may have an effect on the hearing of the subjects (Ward, 1965; Hirsh, 1958; Rajan, 1995). According to literature, monaural exposures might induce a higher impact on hearing levels than binaural exposures (Ward, 1965; Hirsh, 1958; Rajan, 1995). The reason is not clear but it is attributed to either (1) a more vigorously contraction of the middle-ear reflex for binaural exposures; or (2) influences exerted by the crossed efferent pathways.

The magnitude of the differences on the auditory system from monaural vs. binaural exposures is not completely understood. Several researchers have suggested that it might depend on the frequency of the stimulus. Ward (1965) reported from TTS experiments with human subjects exposed to different pure tones, that monaural exposures resulted in a higher TTS particularly at low frequencies; whereas no big differences were observed at other frequencies. Results from Hirsh (1958) also from TTS experiments with humans showed that a exposure to white noise did not result in clear differences between monaural and binaural exposures. However, monaural exposures to a pure tone resulted in a longer recovery period, even though the TTS immediately after the exposure was the same as for the binaural exposure.

### 3.5 Calibration of noise exposures

In both experiments presented in PAPER I and II, the calibration of the noise exposure was verified in five subjects, who were staff at Aalborg University within the age range of the subjects who participated in the formal test. Measurements

		CONT		IMP+CONT	
		L	R	L	R
Subjects	$L_{ear,3min}$	102.5	102.3	98	97.8
	std	0.7	0.7	0.5	0.6
Coupler	$L_{eq,3min}$	107	106.8	103.8	103.2
	std	0.2	0.1	0.1	0.1

Table 3.4: Average SPL and standard deviation (std) from the calibration of the stimuli CONT and IMP+CONT in PAPER II. Results correspond to measurements at the blocked-entrance ear canal of five subjects and in the coupler. Both channels L and R are indicated. Data shown in dB.

were performed with a miniature microphone *Sennheiser KE-4-211-2* at the blocked-entrance ear-canal connected to a frequency-analyzer B&K 2133. Measurements consisted on readings of  $L_{ear}$  for five headphone repositions. Afterwards, the level was measured in a coupler B&K 4153 (IEC 60318-3:1998). Both channels L and R were tested. The headphone was repositioned 10 times for each channel. The calibration was verified weekly in the coupler during the course of the experiment and at the very end.

For the stimulus TONE the average  $L_{ear}$  across subjects was 102.2 dB with a standard deviation of 0.3 dB for the left ear and 0.4 dB for the right ear. The average equivalent SPL in the coupler was 96.8 dB for the left channel and 96.9 dB for the right channel. The standard deviation was 0.11 and 0.07 dB respectively.

Both stimuli CONT and IMP+CONT were presented to the subjects for three minutes. The results of the calibration are shown in table 3.4.





# Chapter 4

## Main results and analysis

### 4.1 Results from PAPER 1

Figure 4.1 shows the effect of the tonal exposure on the characteristics of the fine structures from eight subjects. The analysis of the DPOAE fine-structures is done according to the classification algorithm proposed by Reuter and Hammershøi (2006). The algorithm monitors the ripples of the fine structures, which are characterized by a maximum DPOAE level (peak) located in between two minima (valleys). Changes in the DPOAE fine-structures are studied by plotting the maxima and minima of the ripples during the course of the experiment. A ripple is considered valid (and therefore plotted) if it satisfies two conditions: (1) the ripple height (RH), which is the level difference between the maximum and the mean of the two minima, must be higher than 3 dB; and (2) the maximum of the ripple ( $\text{SNR}_{\max}$ ) must be at least 3 dB above the noise floor. These values are chosen in order to evaluate ripple characteristics with the lowest spread across subjects, and to distinguish true ripples of the fine structures from merely small variations in DPOAE levels.

Figure 4.1 shows that the characteristics of the fine structures are highly individual across subjects. Only subject 9 shows big level differences between peaks and valleys, with the greatest RH of 15 dB, and therefore the maxima and minima are easier to localize. Other subjects (e.g. 1 and 7) do not show pronounced fine structures and the peaks and valleys of the ripples cover a broader frequency range, which makes the detection of the maxima and minima not so evident. Finally, there are subjects with no measurable fine structures (e.g. subject 4).

#### 4.1.1 Group-average DPOAE shift

Figure 4.2 shows the average DPOAE shift across subjects as a function of time and frequency. Data from left and right ears are grouped together. Averages are calculated in intervals of 30 seconds. Intervals are accepted if they include data from at least 10 subjects. The DPOAE shift immediately after the exposure is approximately 5 dB for all frequencies. Most of the recovery occurs within the first 20 minutes after the end of the tonal exposure –as the DPOAE shift drops to about 2 dB for all measured frequencies.

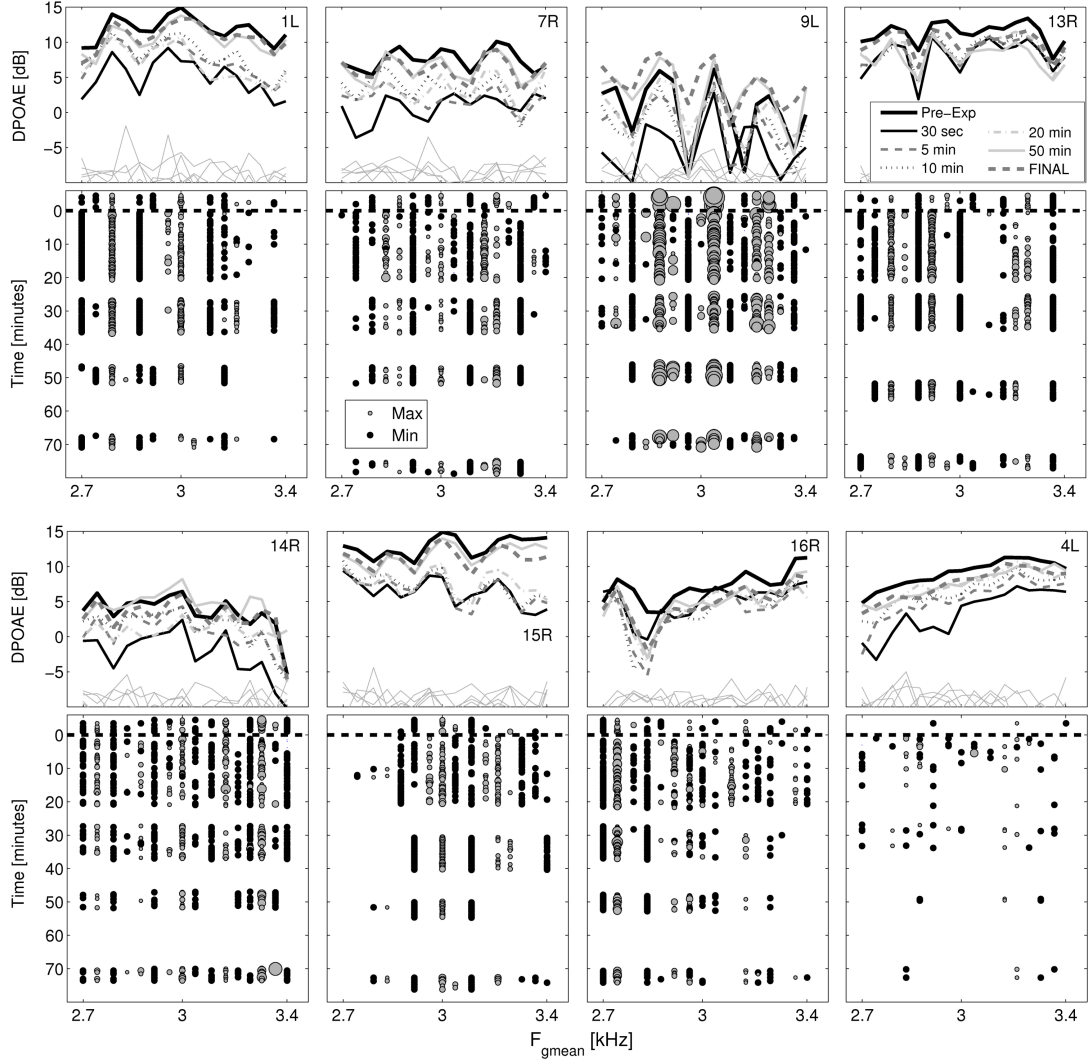


Figure 4.1: DPOAE levels and effects on the fine structures from eight subjects. There are two subplots per subject arranged column-wise. Top: DPOAE levels as a function of frequency at specific instants during the experiment. Bottom: evolution of the maxima and minima of the fine-structure ripples. The abscissa corresponds to the time in minutes after the sound exposure. The horizontal dashed black-line at zero minutes marks the transition between pre and post-exposure measurements. The maxima and minima of the ripples are shown as grey and black circles respectively. The size of the maxima depends on the ripple height, i.e., the bigger the circle the bigger the ripple height.

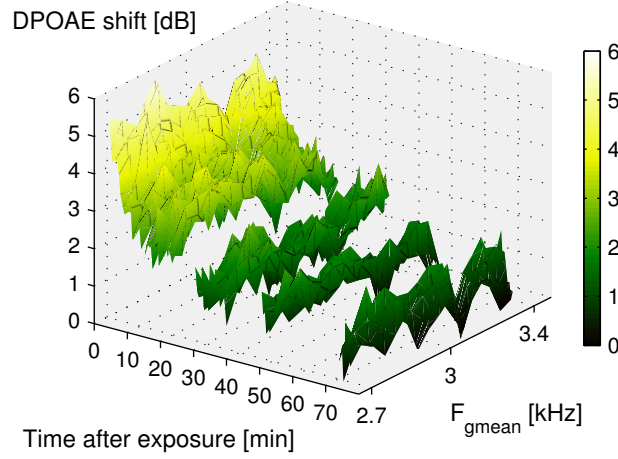


Figure 4.2: Group average DPOAE-shift.

#### 4.1.2 Comparison of pre-exposure and final DPOAE levels

Figure 4.3 shows the group-average DPOAE levels for pre-exposure and final measurements 70 minutes after the sound exposure. It also shows the average noise-floor level in both cases. The difference between the DPOAE level and noise level (the S/N) is between 13 and 15 dB. Such a S/N is considered adequate to obtain reliable DPOAE measurements, in which the stability of DPOAE levels is not influenced by the background noise (Lonsbury-Martin *et al.*, 1993).

A balanced two-way ANOVA analysis is performed to determine whether there are significant statistical differences between pre-exposure and final DPOAE levels. The significance level of the analysis (and all analysis in this thesis) is  $\alpha = .05$ . The factors are: (1) determination time –i.e., pre-exposure/final; and (2) frequency –16 levels. There are no significant differences in the DPOAE levels across frequencies. However, there is a weak statistical difference between pre-exposure and final DPOAE levels ( $F_{(1,975)} = 7.9$ ,  $p = .047$ ). As the p-value is almost the same as the significance level ( $.047 \approx .05$ ), it can be argued that the differences between pre-exposure and final DPOAE levels are negligible.

Next, it is studied whether reinserting the sound probe in the ear canal of the subjects influenced the determination of pre-exposure and final DPOAE levels. Figure 4.4 shows the average standard deviation of DPOAE levels across subjects during the determination of pre-exposure and final levels. The average standard deviation is similar in both cases and it falls within 0.5 – 1.5 dB. Similar results were obtained by Reuter *et al.* (2007), who reported a standard deviation within 1 – 2 dB for the measurements with and without reinsertion of the sound probe. It can also be seen in Figure 4.4 that the average standard deviation is slightly higher for refitted measurements.

Statistical differences in Figure 4.4 are studied by means of a three-way ANOVA analysis. The factors are: (1) probe fitting –i.e., refitted/non-refitted; (2) determination time –i.e., pre-exposure/final; and (3) frequency – 16 levels. There are no significant differences between pre-exposure and final measurements –nor across frequencies. However, the standard deviation of refitted measurements is significantly higher than non-refitted measurements ( $F_{(1,975)} = 75.7$ ,  $p = .001$ ). Finally, there are no significant interactions between the factors.

Figure 4.5 compares the DPOAE levels obtained from repeated measurements

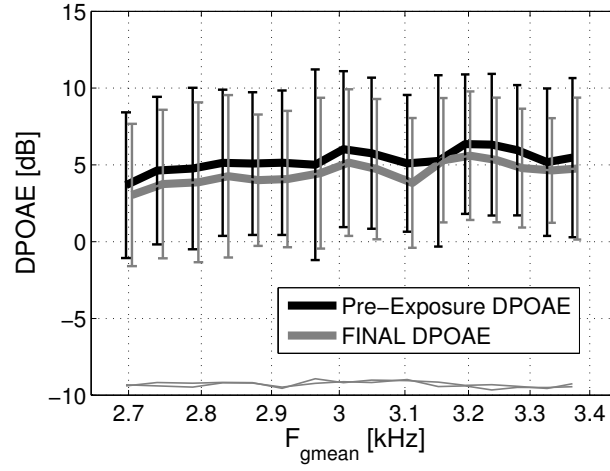


Figure 4.3: Group average DPOAE levels from pre-exposure and final measurements. Final measurements were measured 70 minutes after the sound exposure. Errorbars show the standard deviation across subjects. The thin gray lines at the bottom correspond to the average background noise during the measurements.

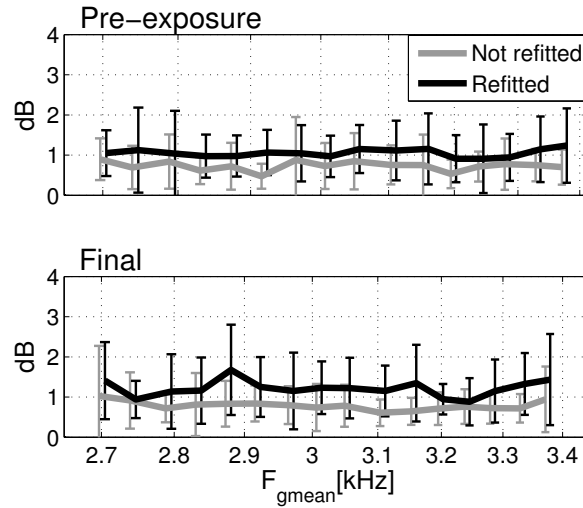


Figure 4.4: Average standard deviation (in decibels) across subjects for refitted and non-refitted DPOAE measurements. The horizontal axis corresponds to the geometric mean of the measured frequency range. Top: pre-exposure; Bottom: final.

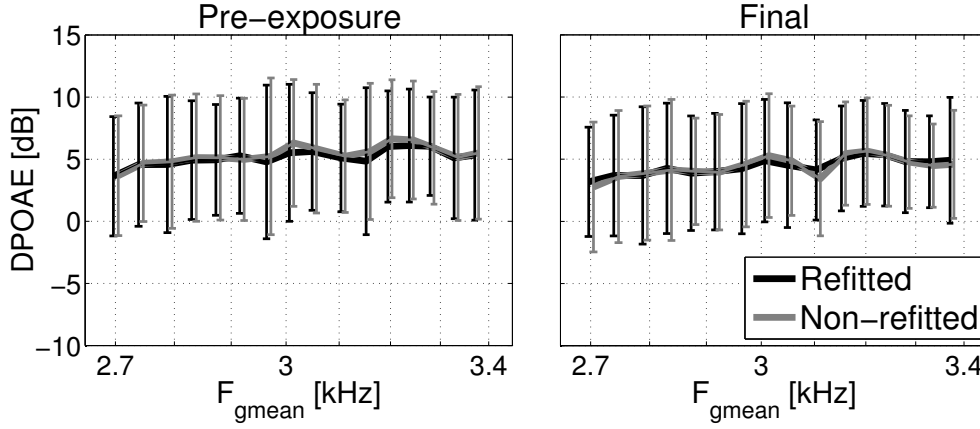


Figure 4.5: Average DPOAE levels for refitted and non-refitted measurements. The horizontal axis corresponds to the geometric mean of the measured frequency range. Left: pre-exposure DPOAE levels; right: final DPOAE levels. The standard deviation across subjects is shown as errorbars.

– with reinsertion of the sound probe and without reinsertion. The comparison is shown for pre-exposure and final DPOAE levels. In both cases the DPOAE levels from refitted and non-refitted measurements are almost identical, as the plots practically overlap each other. A two-way ANOVA analysis is performed to check for statistical differences. The factors are (1) probe fitting (refitted/non-refitted); and (2) frequency (16 levels). No significant differences are found for any of the main effects and their interaction. We conclude therefore that refitting the sound probe in the ear of the subjects caused a slightly higher variation of DPOAE levels during repeated measurements. However, these variations did not result in statistically significant differences during the assessment of DPOAE levels.

#### 4.1.3 Stability of primary levels

Differences in the primary levels  $L_1$  and  $L_2$  might cause the pattern of the fine structures to get shifted along the frequency axis (He and Schmiedt, 1997). The stability of the primary levels during the course of the experiment is depicted in Figure 4.6. The Figure shows the mean of the primaries, averaged across subjects and frequencies. Errorbars show the confidence intervals ( $\alpha = .05$ ). Results are depicted before the sound exposure (*Pre*); at 10-minute intervals during the recovery –except the last interval which takes only 5 minutes; and at the end of the experiment (*Final*). Statistical differences of the primaries during the determination of pre-exposure levels and each of the recovery intervals are investigated by means of a t-test analysis. The results show that there are no significant statistical differences in any of the intervals.

## 4.2 Results from PAPER 2

Figure 4.7 shows the average DPOAE-shift across subjects for the stimuli CONT and IMP+CONT. For each stimulus the results from left and right ears are grouped together. Averages are calculated in intervals of 30 seconds. Intervals are accepted if they include data from at least 10 subjects. It can be seen that the *continuous* noise exposure has a maximum DPOAE shift of approximately 5 dB in the range

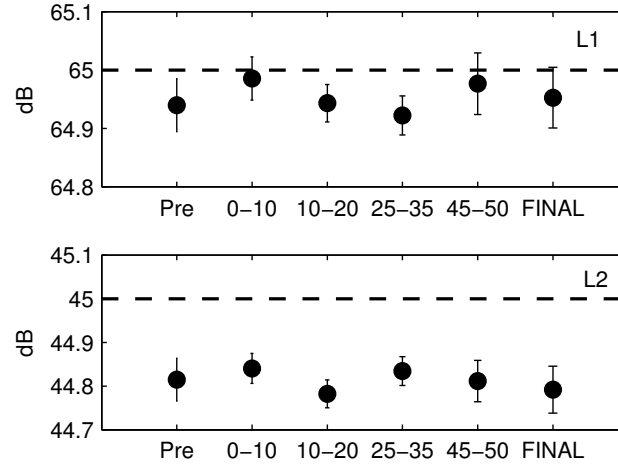


Figure 4.6: Stability of the overall primary levels  $L_1$  and  $L_2$  at different intervals. The errorbars show the confidence interval ( $\alpha = .05$ ). The dashed horizontal lines show target levels of 65 and 45 dB.

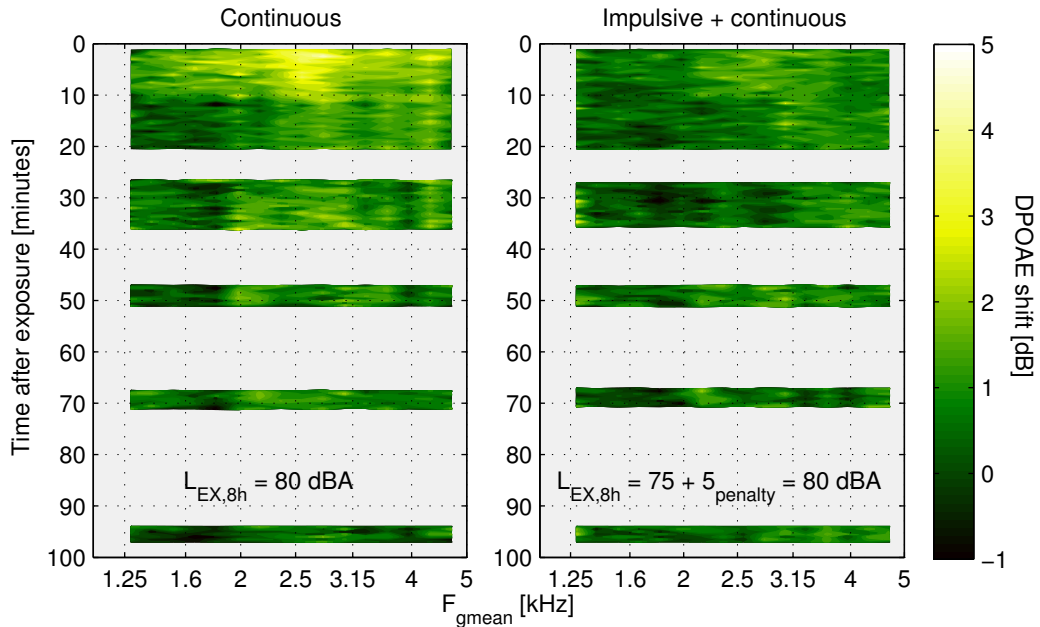


Figure 4.7: Group-average DPOAE-shift from *continuous* and *impulsive+continuous* noise exposures shown as a function of the geometric mean of the primaries and the determination time after exposure.



$2 < F_{gmean} \text{ [kHz]} < 3.15$  during the first 10 minutes of the recovery, whereas the *impulsive+continuous* noise exposure shows no evident DPOAE-shift.

A two-way ANOVA analysis is performed to study whether there are significant differences between the DPOAE levels from stimuli CONT and IMP+CONT throughout the experiment. The factors are: (1) noise stimulus – CONT/IMP+CONT; and (2) frequency (16 levels). The analysis is done in time intervals of 5 and 10 minutes during the recovery. Differences in pre-exposure and final DPOAE levels are also evaluated. The analysis is done by comparing the DPOAE levels from both stimuli measured at the same time interval. Significant differences between the noise stimuli are observed during the first 10 minutes of the recovery ( $F_{1,10528} = 70.9$ ,  $p < .001$ ), which supports the idea that the stimulus CONT had a bigger impact on DPOAE levels than the stimulus IMP+CONT. These differences disappear throughout the rest of the recovery, except at the time interval 45–50 minutes ( $F_{1,5568} = 8.8$ ,  $p < .01$ ). There is a main effect in frequency for all the intervals ( $d.f. = 15$ ,  $F > 29.32$ ,  $p < .001$ ), meaning that the DPOAE shift is significantly higher at some frequencies than others.

#### 4.2.1 Comparison of pre-exposure and final DPOAE levels

The group-average pre-exposure and final DPOAE levels from stimuli CONT and IMP+CONT are shown in Figure 4.8. The Figure also shows the average noise level during the measurements. The S/N across frequencies is between 8 and 14 dB. A balanced four-way ANOVA analysis was performed with the following factors: (1) determination time – pre-exposure/final; (2) fitting effect<sup>1</sup> – refitted/non-refitted; (3) noise stimulus – CONT/IMP+CONT; and (4) frequency (16 levels). There are no significant differences for the following main effects: (1) determination time, meaning that DPOAE levels recovered completely by the end of each exposure; (2) fitting, indicating that reinsertions of the sound probe in the ear canal did not influence the assessment of DPOAE levels; and (3) stimulus. There are however significant differences across frequency ( $F_{15,1981} = 17.12$ ,  $p < .001$ ), which means that subjects have significantly higher DPOAE levels at some frequencies than others. There are no significant interactions between the factors.

Figure 4.9 shows the average standard deviation of DPOAE levels during the determination of pre-exposure and final levels. Results are presented for both stimuli CONT and IMP+CONT. As in the first experiment (section 4.1.2), the average standard deviation is similar in both cases and it falls within 0.5 – 1.5 dB. Also in this case, the average standard deviation is slightly higher for refitted measurements.

Statistical differences in Figure 4.4 are studied by means of a four-way ANOVA analysis. The factors are: (1) probe fitting –i.e., refitted/non-refitted; (2) determination time –i.e., pre-exposure/final; (3) Type of noise –i.e., CONT/IMP+CONT and (4) frequency – 16 levels. There is a significant effect for the probe fitting ( $F_{(1,975)} = 75.7$ ,  $p = .001$ ), indicating that refitted measurements led to a higher deviation of DPOAE levels. No significant differences are found for the rest of the effects and their interactions.

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<sup>1</sup>DPOAE levels from refitted/non-refitted measurements are not shown in Figure 4.8 for clarity reasons

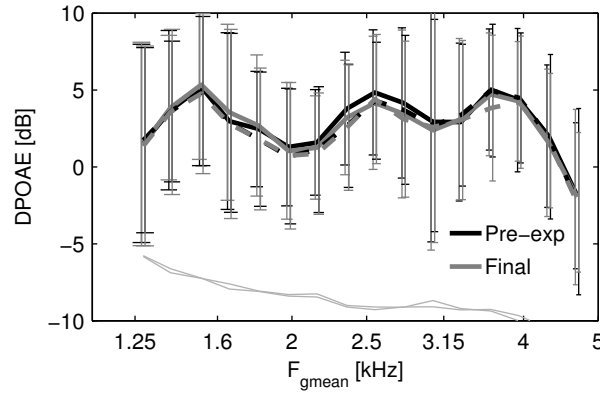


Figure 4.8: Average pre-exposure and final DPOAE levels for the stimuli CONT (continuous lines) and IMP+CONT (dashed lines). Final levels were measured 100 minutes after the exposure. Errorbars show SD across subjects. The thin grey lines at the bottom correspond to the average noise levels.

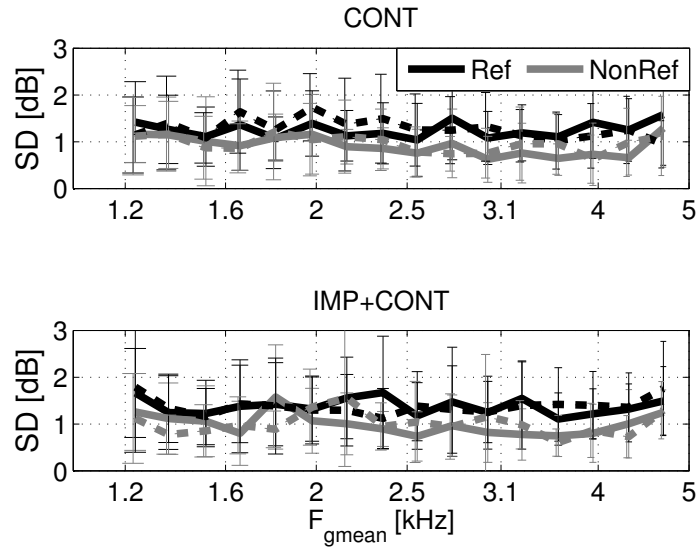


Figure 4.9: Average standard deviation (in decibels) across subjects for refitted and non-refitted DPOAE measurements. The horizontal axis corresponds to the geometric mean of the measured frequency range. Pre-exposure measurements are depicted as continuous lines. Final measurements are depicted as dashed lines. Top: stimulus CONT; Bottom: stimulus IMP+CONT.

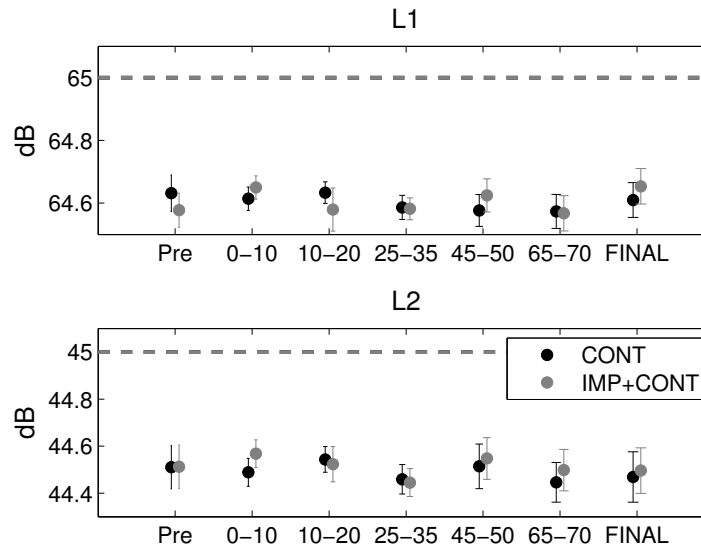


Figure 4.10: Stability of the overall primary levels  $L_1$  and  $L_2$  at different intervals. The errorbars show the confidence interval ( $\alpha = .05$ ). The dashed horizontal lines show target levels of 65 and 45 dB.

### 4.2.2 Stability of primary levels

The stability of the primary levels during the course of the experiment is depicted in Figure 4.10. The figure shows the mean of the primaries, averaged across subjects and frequencies. Errorbars show the confidence intervals ( $\alpha = .05$ ). Results are depicted before the sound exposure (*Pre*); at 10-minute intervals during the recovery—except the last two interval which takes only 5 minutes; and at the end of the experiment (*Final*). Statistical differences of the primaries during the determination of pre-exposure levels and each of the recovery intervals are investigated by means of a t-test analysis. There are no significant statistical differences in any of the intervals.



# Chapter 5

## Overall discussion and conclusion

### 5.1 Influence of sound overexposure on DPOAE fine structures

The results presented in Figure 4.1 show that the characteristics of the DPOAE fine structures are highly individual across subjects. In general, the location of the maxima and minima of the fine structures remained stable throughout the recovery period. For few subjects, the depth (peak-to-valley amplitude) of the fine structure varied considerably after sound overexposure -i.e., some subjects showed an increase in the depth of the fine structures (e.g., subject 13) while others showed a decrease after the exposure (e.g., subject 9)– but in general there were no significant differences in the depth of the fine structures. This variability is in agreement with the results by Reuter *et al.* (2007) and it can be explained according to the two-source model of DPOAE generation. The model considers that the fine structure is caused by constructive and destructive interference of the distortion component  $f_2$  and the reflection component  $2f_1 - f_2$ . Variations of the depth of the fine structures after sound overexposure can be indicative of shifts in the relative amplitude of the two components. Our results indicate that the variation may be more acute for some subjects than others but –on average– there are no significant differences across subjects, which suggests that the two components might be equally affected. This theory could explain why no systematic changes of fine structure could be observed in this study.

The results suggest that the fine structures of the DPOAE might not be a better indicator of early hearing loss than the DPOAE level alone. On the contrary, the variability seen across subjects might complicate the interpretation and comparison of results.

### 5.2 Comparison of temporary changes on DPOAEs

Figure 5.1 shows the average DPOAE-shift across subjects after exposure to the stimulus TONE in PAPER I; and the stimuli CONT and IMP+CONT in PAPER II. The horizontal axis corresponds to the frequency range of the primaries  $f_1$  and  $f_2$ . The frequency range in each subplot is 1.25–5 kHz. This is done in order to facilitate the comparison between the stimuli and to emphasize that the DPOAE recovery from the stimulus TONE was measured in a narrower frequency range. The vertical axis shows the time after the end of the noise exposure in minutes. Results are

presented up to 50 minutes as this is the time period of the recovery that the three stimuli have in common. It can be seen that:

- The stimulus CONT induced a maximum DPOAE-shift of approximately 5 dB in the frequency range  $2 < F_{gmean}$  [kHz] < 3.25. In addition, the DPOAE shift seems to recover mostly within the first 10 minutes after the end of the exposure. Afterwards, the variation of the DPOAE shift seems to be comparable to the standard deviation of repeated measurements depicted in Figure 4.9.
- The stimulus IMP+CONT did not cause an evident DPOAE-shift after the exposure – as the value of the DPOAE shift is similar to the standard deviation of repeated measurements (Figure 4.9).
- The maximum DPOAE-shift from the stimulus TONE is approximately 5 dB in the measured frequency range ( $2697 < F_{gmean}$  [Hz] < 3371), which was 1/3-octave-band centered at 3 kHz. It can also be seen that most of the recovery occurs within the first 20 minutes after the end of the exposure.

The comparison of the three stimuli is not straightforward. First, because of methodological differences in the presentation method. That is, both stimuli CONT and IMP+CONT were presented binaurally, whereas the stimulus TONE was presented monaurally (see section 3.4.6 for an explanation on the influence of the presentation method in the assessment of noise overexposure). Second, the DPOAE-shift from the stimulus TONE was measured in a narrower frequency range than the stimuli CONT and IMP+CONT. From the results it is apparent that the DPOAE shift from the stimulus TONE covered a broader frequency-range than the design allowed for. Unfortunately, it is not possible from our measurements to conclude how much broader in frequency the shift was. Many researchers have reported that narrow-band sound exposures may have an impact on a broader area of DPOAE levels than the one typically seen in TTS experiments (Engdahl and Kemp, 1996; Reuter *et al.*, 2007; Marshall *et al.*, 2001). The same researchers reported that the maximum impact is located 1/2-octave above the sound exposure. Therefore, we consider that for the stimulus TONE the frequency range in our experiment is the most susceptible to show the biggest impact after overexposure, and that other regions not studied here might have shown similar or lower DPOAE-shifts, if any.

Results in Figure 5.1 show that the maximum DPOAE-shift of stimuli CONT and TONE was approximately 5 dB immediately after the exposure but located in a different frequency range. In addition, it can be seen that the DPOAE shift from the stimulus TONE needed more time to recover than for the stimulus CONT. The difference in the frequency specificity can be attributed to the different spectral content of the stimuli. The difference in the recovery period can be interpreted in the two following ways: (1) the longer recovery period of the tonal exposure (TONE) could be due to differences in the presentation method –monaural vs. binaural– as reported by Hirsh (1958); or (2) if we assume that there are no differences induced by the presentation method, the longer recovery period could be a sign of a higher auditory fatigue. The latter argument suggests that tonal exposures might be more hazardous for our hearing than continuous-broadband noise exposures with equal energy. One possible explanation is that for tonal exposures the energy is concentrated in a narrow region of the basilar membrane, which may pose a higher fatigue to the auditory system.

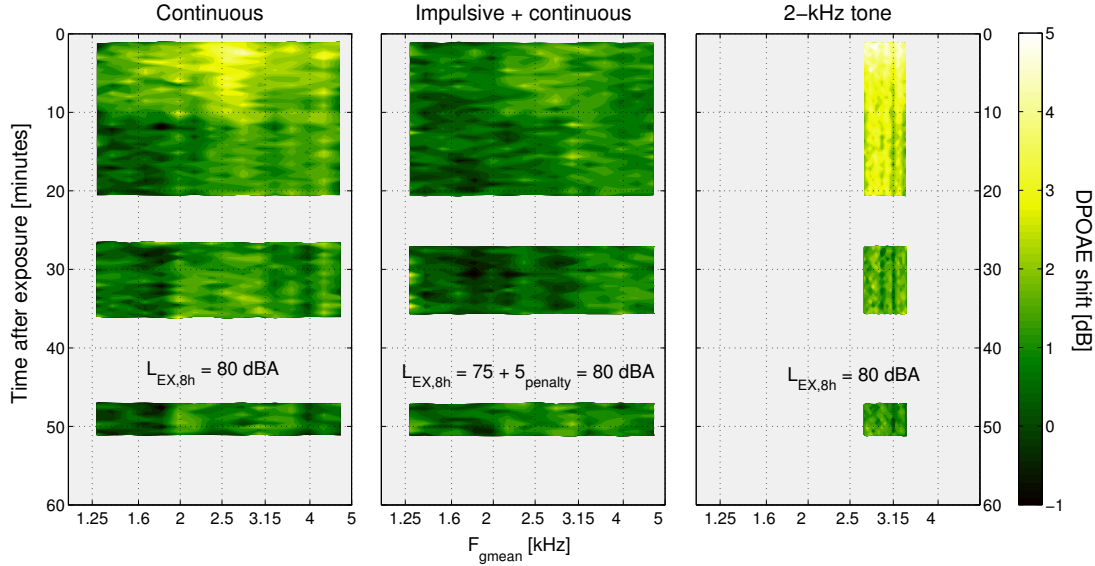


Figure 5.1: Group DPOAE-shift comparison from exposure to stimuli CONT, IMP+CONT and TONE. The horizontal axis corresponds to the measured frequency range (geometric mean). The vertical axis shows the time in minutes after the end of the noise exposure. The blank periods during the recovery correspond to pauses to allow subjects to relax.

The recovery from the stimulus IMP+CONT in Figure 5.1 does not show an evident DPOAE shift. A possible explanation might be that the combination of continuous noise with a series of impulses, presented at a repetition rate of 0.5 impacts per second, had a protective effect on the auditory system. Several researchers have shown that a repetition rate between 0.5–1 impacts per second may pose the highest hazard to hearing, both in humans (Trémolirès and Héту, 1980) and animals (Danielson *et al.*, 1991; Henderson *et al.*, 1991). However, this conclusion is based on data from purely impulsive signals and therefore it cannot be ruled out that the addition of a continuous sound in our experiment instigated some protective effect.

An interesting aspect is that the difference in the maximum DPOAE-shift between the stimuli is approximately +5 dB –the same as the penalty. Whether this is a coincidence still needs to be evaluated. The evaluation could be done by comparing input-output (I/O) functions, in which the magnitude of the DPOAE shift can be plotted as a function of the exposure level. In this way, it would be possible to determine whether there is a linear relation between the magnitude of the DPOAE-shift and the exposure level. In the present study, the fact that the difference in the maximum DPOAE shift is approximately +5 dB suggests that the results can be better explained on an energy basis, in which the magnitude of the DPOAE-shift may increase at a rate of approximately 1 dB per dB in the exposure level. This is in agreement with the results by Eddins *et al.* (1999), in which a group of chinchillas was continuously exposed to an octave-band noise centered at 4 kHz for a total of 42 days, 6 days at each of seven exposure levels. The exposure level increased in 8-dB steps from 48 to 96 dB SPL. DPOAE input-output (I/O) functions were measured at octave intervals over a range of primary tone  $f_2$  frequencies between 1.2 and 9.6 kHz. The primaries were presented at equal level ( $L_1 = L_2$ ) and with a  $f_2/f_1$  ratio of 1.2. DPOAE I/O functions were measured in 5-dB increments from 0 to 80 dB SPL. Measurements were obtained (1) pre-exposure, (2) during days 3-6 of each 6-day exposure, and (3) 4 weeks after the final exposure. The maximum DPOAE shift was



observed within the octave-band of the noise exposure (3.4 – 6.8 kHz). The combination of primaries that revealed that biggest DPOAE-shift was  $L_1 = L_2 = 75$  dB SPL. For this set of primaries the DPOAE shift increased at a ratio of 1.3 dB for every dB increase in noise level up to approximately 75 dB SPL. Thus, the ratio observed in our experiment is comparable to the ratio observed by Eddins *et al.* (1999).

DPOAE measurements in PAPER II were performed with low frequency resolution, insufficient to monitor the fine structures of the DPOAE. For this reason, it is not possible to confirm whether more complex noise exposures –e.g., CONT and IMP+CONT– induced a similar effect on the DPOAE fine structures as the exposure TONE in PAPER I. The main reason for not monitoring the DPOAE fine structures in PAPER II is that the experimental protocol for DPOAE assessment only allows a maximum of 16 pairs of primaries. As results from PAPER I showed no systematic changes in the characteristics of the DPOAE fine structures after a tonal overexposure, it was decided to disregard the assessment of the fine structures in PAPER II in benefit of DPOAE measurements on a broader frequency range.

In the present study, changes in the properties of DPOAEs were evaluated by monitoring the cubic distortion product  $2f_1 - f_2$ . However, several researchers have reported that the quadratic distortion product  $f_2 - f_1$  may be more sensitive than  $2f_1 - f_2$  to evaluate changes in the inner ear after noise overexposure. Animal studies have shown a bigger amplitude reduction in the component  $f_2 - f_1$  than  $2f_1 - f_2$  after ipsilateral and contralateral acoustic stimulation (Chang and Norton, 1997; Kirk and Johnstone, 1993). These findings suggest an adaptive process within the cochlea that may be modulated by efferent stimulation. Similar findings have been reported for human subjects after contralateral stimulation (Wittekindt *et al.*, 2009). The authors attributed the higher sensitivity of  $f_2 - f_1$  to a shift of the operating state and/or a change in the gain of the cochlear amplification due to contralateral induced efferent modulation of the OHCs properties. Unfortunately, human data are rare due to the low amplitude of the  $f_2 - f_1$  tone – which makes it almost immeasurable in human subjects (Meinke and Stagner, 2005).

### 5.2.1 Comparison of recovery functions

Next, a recovery function is obtained for the three noise exposures. The recovery function models the DPOAE shift averaged across subjects and frequencies.

The recovery of the DPOAE shift after exposure to the stimulus TONE in the measured frequency range can be modeled with two exponential functions of coefficients:

$$\text{TONE: } y = 1.63 \cdot e^{-t/3.33} + 3.45 \cdot e^{-t/47.6} \quad (5.1)$$

This fitting yields a *coefficient of determination*<sup>1</sup>  $R^2$  of 0.77.

As the recovery function from the stimulus TONE is obtained in a frequency range of approximately 1/3-octave band, we select a similar frequency range for exposures to CONT and IMP+CONT. The frequency range corresponds to  $2563 < F_{gmean} [\text{Hz}] < 3049$  and it is the range that shows the maximum DPOAE-shift from the stimulus CONT. In this way, the recovery functions are:

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<sup>1</sup>The coefficient of determination  $R^2$  provides a measure of how well future outcomes are likely to be predicted by the model. The values can vary from 0 to 1 –i.e., from poor to perfect.

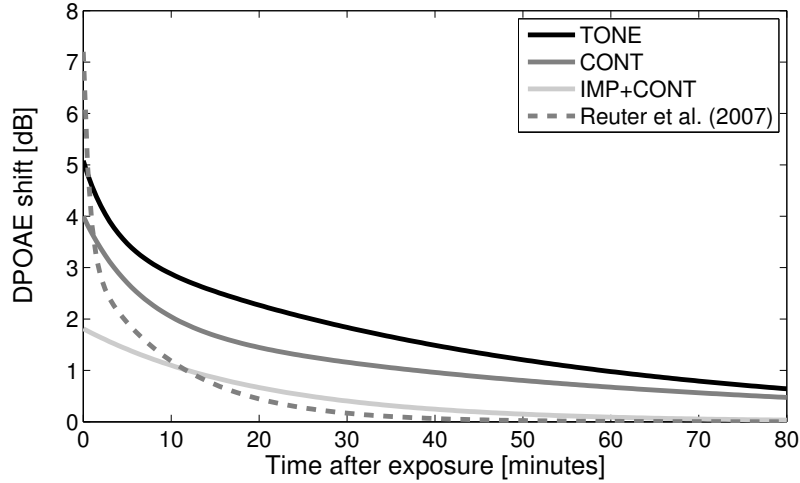


Figure 5.2: Recovery functions from stimuli TONE, CONT and IMP+CONT. The recovery function obtained by Reuter *et al.* (2007) is also shown. The ordinate shows the DPOAE shift and the abscissa corresponds to the time in minutes after the sound exposure.

$$\text{CONT: } y = 2.06 \cdot e^{-t/6.28} + 1.93 \cdot e^{-t/56.8} \quad (R^2 = 0.74) \quad (5.2)$$

$$\text{IMP+CONT: } y = 1.82 \cdot e^{-t/20} \quad (R^2 = 0.55) \quad (5.3)$$

Which are modeled with two exponential functions for the exposure CONT, and one for IMP+CONT.

The recovery functions of the exposures TONE, CONT and IMP+CONT are depicted in Figure 5.2. For the stimulus CONT, the DPOAE shift falls to approximately 1.5 dB within the period 30–40 minutes after exposure. Following values below 1.5 dB are within the standard deviation of repeated measurements (Figure 4.9). For the stimulus IMP+CONT, the DPOAE shift falls to approximately 1 dB (the standard deviation of repeated measurements shown in Figure 4.4) around 70 minutes post-exposure. This means that the stimulus TONE induced the biggest DPOAE-shift and the longest recovery period.

Figure 5.2 also includes the recovery function obtained by Reuter *et al.* (2007) for comparison ( $y = 4.06 \cdot e^{-t/0.7} + 3.14 \cdot e^{-t/10.24}$ ). In their study, 39 normal-hearing subjects were monaurally exposed for three minutes to a pure tone of 1 kHz normalized to  $L_{EX,8h} = 85$  dB. DPOAEs were measured with an ascending sweep in the range  $903 \leq f_2$  [Hz]  $\leq 2295$  that took almost 7 minutes to complete. It can be seen that the DPOAE-shift immediately after exposure reaches a value of approximately 7 dB. The differences in the maximum DPOAE-shift between our results and the ones by Reuter *et al.* (2007) can be attributed to the higher exposure level used in their experiment. However, the recovery function obtained by Reuter *et al.* (2007) seems to return to 0 dB within the first 30 minutes of the recovery period. *How is it possible that a more severe exposure took less time to recover?* One reason could be that the sound exposure used by Reuter *et al.* (2007) had a shorter duration than the one in our experiment –3 vs. 10 minutes. Another explanation could be that our methodological protocol for DPOAE assessment might have influenced the recovery of DPOAE levels. In the experiment by Eddins *et al.* (1999), in which the authors exposed groups of chinchillas to different exposure

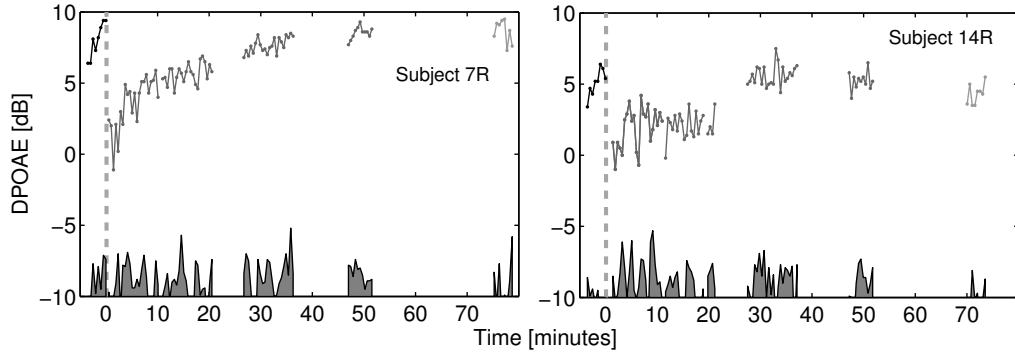


Figure 5.3: DPOAE levels as a function of time for subjects 7 (left) and 14 (right) at  $2f_1 - f_2 = 2356$  Hz ( $F_{gmean} = 3328$  Hz). The ordinate shows DPOAE levels and the abscissa the time in which the DPOAE was measured. The vertical dashed-line at zero minutes marks the transition between pre and post-exposure measurements. The background noise is also shown (grey area).

levels for six days (previously described in section 5.2), a decrease in the DPOAE level could be seen already at 50 dB SPL. In our study, DPOAE measurements were performed with high time resolution –e.g., data corresponding to the same DP frequency were obtained every 28 seconds– and primary levels  $L_1/L_2 = 65/45$  dB. According to the results by Eddins *et al.* (1999), we hypothesize that the presence of external stimuli (primaries  $f_1$  and  $f_2$ ), presented at such a high rate of assessment and relatively high levels, might have inhibited the recovery of OHC function and thereby DPOAE levels. In addition, this effect could be partly responsible for the highest DPOAE-shift observed for the stimulus TONE throughout the recovery period; as in this case, DPOAE measurements were performed with high time-resolution in a narrow area of the basilar membrane, which could have induced a higher fatigue on that particular area.

The enervating effect of the methodological protocol would also explain the *abnormal* recovery pattern of DPOAE levels observed in some subjects. As an example, Figure 5.3 compares the results of two subjects after exposure to the stimulus TONE (PAPER I). The Figure shows the recovery of DPOAE levels as a function of time during the course of the experiment at one particular DP frequency ( $2f_1 - f_2$ ). It can be seen that subject 7 shows a gradual recovery of DPOAE levels –which is a general trend for most of the subjects in the experiment. Subject 14, on the contrary, does not show an evident recovery during the first 20 minutes. However, the DPOAE levels of this subject seem to recover drastically after the first pause, which suggests that cessation of DPOAE assessment favored the recovery of DPOAE levels.

It is not possible to conclude whether the recovery data from subjects with a gradual recovery –e.g., subject 7 in Figure 5.3– were also influenced by the methodological protocol. Perhaps DPOAE measurements at longer intervals of time would have facilitated the recovery of DPOAE levels and thereby obtain a recovery function similar to the one reported by Reuter *et al.* (2007). Our decision for monitoring DPOAEs with high time-resolution represents an attempt to obtain detailed information of the recovery properties of DPOAEs. However, in view of the results, measurements at longer intervals of time might be more appropriate to monitor the true recovery of DPOAE levels.

### 5.2.2 Estimation of maximum TTS

Pure-tone audiometries were performed before the noise exposures and by the end of the experimental sessions. The results averaged across subjects for left and right ears (shown in the corresponding papers) indicate that there are no significant differences in the auditory thresholds of the participants, which means that hearing levels were able to recover within the experimental session. Unfortunately, it is not possible to know the characteristics of the TTS induced by the stimuli TONE, CONT and IMP+CONT –i.e., the maximum TTS, the most affected frequency range and the temporal characteristics of the recovery. We decided to only monitor DPOAEs during the recovery period in an attempt to obtain detailed information of the temporal characteristics of their recovery; especially in the early part. This was particularly desired in order to continuously follow the evolution of the fine structures in PAPER I.

Several researchers have compared the characteristics of TTS and DPOAE-shift (e.g., Sutton *et al.*, 1994; Marshall *et al.*, 2001). These studies have shown that the magnitude of the TTS is generally higher than the DPOAE-shift although the temporal characteristics of their recovery are similar. Of especial attention are the results by Marshall *et al.* (2001), who in a similar experiment to the one presented in PAPER I, studied the relationship between TTS and DPOAE amplitude shifts following a 10-minute monaural noise-exposure in 14 subjects (105 dB SPL half-octave narrow-band noise at 1.414 kHz center frequency). Hearing thresholds and DPOAEs were measured at 2 kHz (1/2-octave above the sound exposure) with measurements interleaved across the same recovery function. The average DPOAE shift for the group at approximately two minutes post-exposure was 5.0 dB ( $L_1/L_2 = 65/45$  dB SPL) and the mean TTS was 11.6 dB. As in our experiment the stimulus TONE also resulted in a DPOAE shift of approximately 5 dB, we expect that the TTS of our subjects may have reached a similar value to the one obtained by Marshall *et al.* (2001).

## 5.3 Implications for noise standards

Figure 5.1 on page 67 shows that both stimuli CONT and TONE induced a maximum DPOAE shift of approximately 5 dB; whereas there is no evident shift due to the stimulus IMP+CONT. Therefore, the +5 dB penalty failed in predicting that the DPOAE shift from the stimulus IMP+CONT normalized to an exposure level of  $75 + 5_{dBpenalty} = 80$  dBA, would be similar to that from the stimuli CONT and TONE normalized to 80 dBA. Furthermore, the combination of impulsive and continuous noise did not pose a bigger impact on DPOAE levels. At least not higher than what would be expected according to the EEH.

We conclude, according to Figure 5.1 and the discussion in section 5.2, that the +5 dB penalty for tonality and impulsiveness recommended by ISO 1999:1990 might be more appropriate for tonal exposures –as tonal exposures may induce a longer recovery period. The penalty, however, might overestimate the auditory hazard for the case of low-level impulse noise, at least for impulses with peak levels below 117 dBC –as the one used in our experiment. It is not possible to generalize this conclusion to impulses with higher peak levels. More empirical data from laboratory and field studies are necessary on this issue.

Whether this conclusion can be extrapolated to other types of impulsive noise still needs to be evaluated. Nevertheless, similar results may be expected for im-

pulses not exceeding 117 dBC peak SPL with either (1) different spectral content; or (2) presented at lower repetition rates. In the first case, impulses with a dominant spectral content in the mid frequency range (2–5 kHz), like the ones used in our experiment, are considered to be more hazardous to hearing than impulses with spectral energy concentrated at lower or higher frequencies. This statement is based on the results by Patterson *et al.* (1993), who exposed groups of chinchillas to narrow-band impulses of different spectral content. They concluded, based on audiometric measurements and estimations of OHC loss, that impulses with spectral energy in the mid frequency range produced the highest damage. In the second case, human and animal data from both TTS and PTS studies have shown that a repetition rate of one impact per second may pose the highest auditory hazard (Trémolirès and Héту, 1980; Danielson *et al.*, 1991; Henderson *et al.*, 1991). This is because impacts within this range are not attenuated by the middle-ear reflex and pose the highest rate of acoustic stress to the inner ear. The repetition rate of 0.5 impulses per second used in this study is approximately the same as the one considered the most harmful, and therefore, lower repetition rates might not pose a higher hazard.

Results from the present study suggest that the risk of NIHL from impulsive exposures with peak levels below 117 dBC may be reasonably predicted according to the EEH. This is in agreement with the recent study by Surovov *et al.* (2001), in which they suggested that the EEH might be a good predictor of hearing loss for impulsive environments with low peak-levels. In their study, the authors compared the hearing levels of forge hammering workers in two different industrial plants, and studied whether the degree of hearing loss could be predicted according to ISO 1999:1990. The equivalent A-weighted levels in both plants were approximately the same, but the degree of impulsiveness varied significantly. The degree of hearing loss of workers exposed to low-level impulses (113-120 dBC) could be predicted according to the standard; whereas the group exposed to higher peak levels (115-143 dBC) showed a significantly higher hearing loss. These results indicate that the peak level of the impulse may be a critical factor in the development of hearing loss. The authors concluded that the +5 dB penalty may be more suitable for noises with peak levels above 120 dBC. Our results agree with their conclusion and extend their findings also to DPOAEs.

Finally, the Danish noise standard Arbejdstilsynet establishes that a +5 dB penalty must be added when measuring noises of impulsive character with peak levels higher than 115 dBC or dBA more than once per minute. Our results suggest that an action level of 115 dBC (or dBA) may be overprotective in many situations.

## 5.4 Future Work

The next step for the experiment in PAPER II would be to include a third stimulus IMP+CONT normalized to an exposure level  $L_{EX,8h}$  of 80 dBA – with no penalization for impulsiveness. Our results suggest that such a stimulus would be innocuous for the hearing of the subjects participating in the experiment. If this third stimulus showed the same effects on DPOAE levels as the stimulus CONT, then the conclusion that the +5 dB penalty was excessive would be strengthened. With our current results, it is not possible to know what differences might arise from *continuous* and *impulsive+continuous* noise of equivalent energy levels.

Further work may also include a purely impulsive signal IMP normalized to

$L_{EX,8h} = 80$  dBA. In this way, it would be possible to study whether there are differences in the fatigue process between purely impulsive sounds and the combination of impulsive and continuous noise in our experiment had a protective effect on the recovery of DPOAEs.

Finally, future work may also study whether the characteristics of the DPOAE fine structures are affected after exposure to more complex types of noise. If exposures to stimuli CONT, IMP and IMP+CONT –presented all at  $L_{EX,8h}$  of 80 dBA– do not result in statistical changes in the characteristics of the fine structures, then the conclusion that the risk of NIHL from low-level impulses may be predicted on an equal energy basis would be reinforced.





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# PAPERS





# Recovery of distortion product otoacoustic emissions after a 2-kHz monaural sound-exposure in humans: effects on fine structures

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A better understanding of the vulnerability of the fine structures of distortion product otoacoustic emissions (DPOAEs) after acoustic overexposure may improve the knowledge about DPOAE generation, cochlear damage, and lead to more efficient diagnostic tools. It is studied whether the DPOAE fine structures of 16 normal-hearing human subjects are systematically affected after a moderate monaural sound-exposure of 10 minutes to a 2 kHz tone normalized to an exposure level  $L_{EX,sh}$  of 80 dBA. DPOAEs were measured before and in the following 70 minutes after the exposure. The experimental protocol allowed measurements with high time and frequency resolution in a 1/3 octave-band centered at 3 kHz. On average, DPOAE levels were reduced approximately 5 dB in the entire measured frequency-range. Statistically significant differences in pre and post-exposure DPOAE levels were observed up to 70 minutes after the end of the sound exposure. The results show that the effects on fine structures are highly individual and no systematic change was observed.

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## I. INTRODUCTION

Distortion-product otoacoustic emissions (DPOAEs) are widely used to evaluate risk of hearing loss from noise overexposure. However, one of the limitations is that the generation mechanisms of DPOAEs are not yet fully understood and therefore researchers are still investigating how changes observed in DPOAE levels may relate to changes in absolute hearing levels, as well as the principles behind DPOAE measurements that might lead to a future standardized method for the assessment of cochlear health. Hence, more research is necessary before DPOAEs can be fully applied for clinical purposes or hearing conservation programs (Marshall *et al.*, 2001).

One particular question for which there is not yet clear evidence refers to the origins of the fine structures of the DPOAE and whether their evaluation might provide further information about the vulnerability of the cochlea after acoustic overexposure. When DPOAE measurements are performed with small steps in frequency, the  $2f_1 - f_2$  acoustic distortion-product can show a particular pattern characterized by a series of amplitude peaks and valleys across frequency with peak-to-valley amplitude ratios as great as 20 dB (Gaskill and Brown, 1990b; Heitmann *et al.*, 1996; Reuter and Hammershøi, 2006) and a periodicity of 3/32 octaves (He and Schmiedt, 1993). This distinct characteristic in DPOAE levels with small frequency intervals is known as fine structures. The reason for the fine structures is not completely understood but it is widely accepted that they are the result of the interaction of two components (Shaffer *et al.*, 2003): (1) the distortion component generated in the overlap region of the two-stimulus tones – which approximates to the

region of  $f_2$ ; and (2) the reflection component generated at the  $2f_1 - f_2$  place. The DPOAE measured in the ear canal is the vector sum of contributions arising from these two regions. The two components interfere with each other either constructively or destructively, producing as much as +6 dB higher levels at some frequencies, and almost canceling at other frequencies, in a distinct periodic pattern.

DPOAE fine structure might be observable whenever the acoustic distortion-product is measurable, and its sharpness, defined as the peak-to-peak frequency distance, is independent of age and hearing loss (He and Schmiedt, 1996). Some authors suggest that the presence of fine structures is a property of the healthy ear and therefore their evaluation may reveal more information about the state of hearing than the DPOAE level alone – e.g., the fine structures reappear after a sudden hearing loss (Mauermann *et al.*, 1999b); and are reduced after aspirin consumption (Rao *et al.*, 1996). Other authors claim that the high variability usually seen in the pattern of the fine structures complicates the interpretation and comparison of results across subjects (Shaffer, 2008; Heitmann *et al.*, 1996). Particularly critical is that the pattern of the fine structures get shifted along the frequency axis when the primary levels  $L_1$  and  $L_2$  are varied (He and Schmiedt, 1997) or the frequency ratio  $f_2/f_1$  is changed (Mauermann *et al.*, 1999; He and Schmiedt, 1997). This stimulus-dependent frequency shift complicates the comparison of DPOAE growth functions with audiometric thresholds, and it might be responsible for the lack of correlation between DPOAEs and hearing thresholds reported by many researchers (Shaffer *et al.*, 2003; Heitmann *et al.*, 1996). Several methods exist to avoid the detection of the fine structures by removing the contribution from the reflection component (Kalluri and Shera, 2001; Long *et al.*, 2008; Dhar and Shaffer, 2004). However, it is not clear yet whether these methods may

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improve the ability of DPOAEs to evaluate hearing loss and therefore further research is necessary before they can be applied to assess the status of hearing. One reason is that the methods require that the generation ( $f_2$ ) and the reflection component ( $2f_1 - f_2$ ) are linearly related, and currently there is a lack of data showing whether this is true, both under normal conditions and under adverse (overexposed) conditions.

Two studies have investigated whether there are systematic changes in the DPOAE fine structures of humans after acoustic overexposure (Engdahl and Kemp, 1996; Reuter *et al.*, 2007). Both used a monaural narrow-band sound-exposure and monitored the fine structures at specific times during the recovery. Their results show that the most affected frequency range was located 1/2-octave above the exposure—similar to the 1/2-octave shift seen in the temporary threshold shift (TTS). However, they reported different effects in the fine structures. Engdahl and Kemp (1996) found for their two subjects that the maximum to minimum ratio of the fine structure decreased, and the whole pattern shifted toward lower frequencies after the exposure. Reuter *et al.* (2007) found for their 16 subjects that the effects were highly individual and no systematic change was observed—i.e., some subjects showed an increase in the depth of the fine structures while others showed a decrease, with no systematic shift in frequency. Results by Reuter *et al.* (2007) may have been influenced by the methodological protocol for DPOAE assessment. That is, DPOAEs were measured with an ascending sweep in the range  $903 \leq f_2$  [Hz]  $\leq 2295$  that took almost 7 minutes to complete. Thus, comparisons of DPOAE levels across frequency might be biased by the time-shift during assessment—as they might correspond to a different state of the recovery process in the inner ear.

The scope of the present experiment is to study whether the fine structures of the DPOAE are systematically affected after a moderate monaural sound-exposure in human subjects. A systematic change might indicate that one of the components of DPOAE generation is more sensitive to noise overexposure—which means that the components are not linearly related. Measurements were performed in a narrow frequency range with high time and frequency resolution, in an attempt to provide a detailed description of the fine structures during the recovery. The experiment was approved by the Danish National Ethical Committee on Biomedical Research Ethics.

## II. MATERIALS AND METHODS

### A. Subjects

In total, 16 subjects (eight males and eight females) participated in the experiment. The average age was  $24.1 \pm 2.3$  years. Subjects had hearing thresholds below 20 dBHL from 250 Hz to 8 kHz measured in octave intervals; a middle-ear pressure within  $\pm 50$  daPa; and an active stapedius reflex. None of the subjects reported known incidents of excessive noise exposures or abnormal reactions to noise, e.g. annoyance or headaches. All sub-

jects were students at Aalborg University and they were remunerated for their participation.

### B. Sound exposure

A pure tone of 2 kHz was presented monaurally for 10 minutes via a pair of headphones model Sennheiser HDA 200. The exposure level was normalized to an 8-hour working-day of  $L_{EX,8h} = 80$  dBA (ISO 1999:1990). This level corresponds to an  $L_{ear,10min} = 102.2$  dB measured at the blocked-entrance ear canal (ISO 11904-1:2002). The standard deviation across subjects was  $\pm 0.4$  dB in the worst ear. The exposure frequency of 2 kHz was chosen in order to avoid DPOAE measurements with poor S/N ratio in the low frequency-range due to (1) a higher background noise mainly attributed to physiological noise from the subject; and (2) low DPOAE levels across subjects (Reuter and Hammershøi, 2006). The exposure duration of 10 minutes was a compromise between avoiding (1) the *two-minute bounce* after intense tonal/narrow-band exposures of less than four minutes observed in measurements of TTS (Quaranta *et al.*, 1998) and DPOAEs (Kirk and Patuzzi, 1997); and (2) long experimental sessions that might be exhausting for subjects. Subjects were instructed to remove the headphones at any time if they felt any discomfort during the sound exposure. None of them did.

### C. Instrumentation

#### 1. Pure-tone audiometry and middle-ear test

Pure-tone audiometries were measured using the commercial audiometer *Madsen Orbiter 922* and the audiometric headphones *Telephonic TDH39*. The left ear was tested first. Hearing thresholds were determined in a frequency range from 250 Hz to 8 kHz with a frequency resolution of one octave by using the ascending method, which complies with the norms for automatic audiometries (ISO-8253:1989). Tympanometric and stapedius-reflex tests were performed with the *Interacoustics impedance audiometer AT235*.

#### 2. Assessment of DPOAE

DPOAEs were measured with the ILO96 Research-system from Otodynamics, using the *DPOAE macro* operating mode. This mode is able to perform measurements in loop periods with a maximum of 16 user-defined pairs of primaries  $f_1$  and  $f_2$ . Individual macros were programmed for each subject. The only difference between macros was the presentation order of the primaries, which was counter-balanced across subjects by means of a Latin-square design.

According to the 1/2-octave shift, it was expected that a tonal exposure of 2 kHz would affect mostly the cochlear region around 3 kHz (Engdahl and Kemp, 1996; Reuter *et al.*, 2007; Marshall *et al.*, 2001). Therefore,

DPOAEs were measured across a frequency range of approximately 1/3-octave centered at 3 kHz (geometric mean of the primaries). This frequency range was covered by 16 equally-spaced pairs of primaries with a frequency resolution of 48 Hz. The averaging time per each pair of primaries presented was set to 1.3 seconds. With this configuration the ILO96 takes 28 seconds to measure the 16 pairs of primaries in the frequency range.

DPOAE measurements were performed using a ratio of  $f_2/f_1 = 1.22$  and fixed primary levels of  $L_1/L_2 = 65/45$  dB. This choice is a compromise between: (1) high sensitivity to detect small changes after noise overexposure –i.e., maximum shifts in DPOAE levels can be obtained when lowering  $L_2$  compared to  $L_1$  (Gaskill and Brown, 1990a; Hauser and Probst, 1991; Whitehead *et al.*, 1995; Marshall *et al.*, 2001); (2) measurable DPOAEs: Whitehead *et al.* (1995) measured DPOAEs for various primary-level combinations at different frequencies and reported that 65/45 dB showed relatively high level DPOAEs for all tested frequencies; and (3) detect the presence of fine structures: measurements with varying equal primary levels ( $L_1 = L_2 = 40$  to 70 dB) have shown a flattening of the fine structure for some subjects at high levels (He and Schmiedt, 1993; Heitmann *et al.*, 1996).

The presence of spontaneous otoacoustic emissions (SOAEs) in the ear of the subjects was not measured.

#### D. Sessions and experimental protocol

The experiment took place at the Section of Acoustics of Aalborg University and it was divided in two sessions – session 1 and 2. Both sessions were conducted in a double-walled, sound-isolated audiometry-chamber, which complies with the background noise requirements stated in ISO 8253-1:1989.

Session 1 was a screening session with the following purposes: (1) to inform subjects about the experiment; (2) to check whether subjects were otologically normal; and to verify that (3) subjects felt comfortable during DPOAE measurements; (4) the sound probe fitted easily in their ear canal; and (5) the primary levels for the test were correctly presented. Subjects listened shortly to the sound exposure. They were allowed to decline participation after this (and at any other point in time). The duration of session 1 was approximately one hour.

The sound exposure and the assessment of DPOAE recovery took place in session 2, which lasted approximately two hours. There was approximately one week in between sessions 1 and 2. Subjects reported that they were not exposed to high-level noises at least one day before session 2. The order and duration of the measurements is depicted in figure 1 and it consisted of:

1. **Audiometry** ( $\approx 20$  minutes). Both ears were tested.
2. **DPOAE** ( $\approx 10$  minutes). Only the ear under measurement was tested to determine pre-exposure DPOAE levels.

#### 3. Monaural sound-exposure (10 minutes)

4. **DPOAE** (50 minutes). The recovery of DPOAE is measured in three time intervals (called A, B and C in figure 1) with durations of 20, 10 and 5 minutes respectively. There were two pauses of 5 and 10 minutes respectively to allow subjects to relax. Pauses took place 20 and 35 minutes after the exposure. Subjects stayed in the room unless they asked to go to the bathroom. Only few did.

5. **Audiometry** ( $\approx 20$  minutes). Both ears were tested.

6. **DPOAE** ( $\approx 10$  minutes). The final DPOAE level was determined in the test ear.

#### E. Measurement procedure

Subjects were comfortably seated on a chair with a head rest and remained calm and still. The ear under measurement was randomly chosen and balanced across subjects. Prior to each measurement, the ILO96 verifies the correct fitting of the sound probe in the ear canal by a *checkfit* procedure. During the checkfit two broadband-click stimuli are alternately delivered by the two output transducers of the sound probe. The checkfit result is stored in an array and used during data collection to balance and normalize the two stimuli levels. All spectrum analyses are done by the ILO96, which performs a fast Fourier transform FFT with a frequency resolution of 12.2 Hz. The noise is estimated from the ten Fourier components nearest to but not including the  $2f_1 - f_2$  frequency. The noise is represented as all levels within two standard deviations of the background noise, i.e., the limits of the 95 % confidence region.

##### a. Determination of the pre-exposure DPOAE level

The pre-exposure DPOAE level is obtained as the average of eight repeated measurements. DPOAE levels below the noise floor are not included in the analysis. For four consecutive measurements the sound probe was reinserted in the ear canal of the subject before each measurement (so-called *refitted*). For the remaining four measurements the sound probe was not reinserted (*non-refitted*). The presentation order of refitted and non-refitted measurements was balanced across subjects. A *checkfit* was performed every time the sound probe is reinserted.

##### b. Assessment of the DPOAE recovery

The headphones were removed immediately after the sound exposure, the sound probe was placed in the ear canal of the subject, and a checkfit was performed. On average, DPOAE collection started  $30 \pm 13$  seconds after the end of the sound exposure, except for one subject,

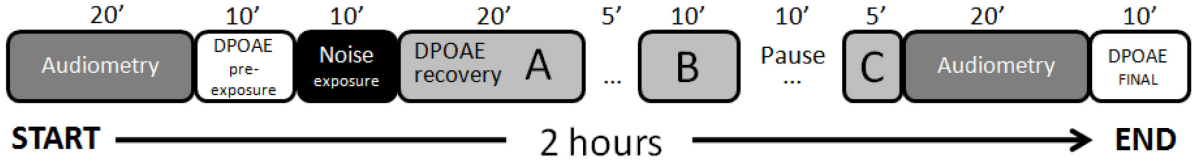


FIG. 1. Order of measurements during session 2. The duration of each measurement is shown in minutes.

for whom it started 1 minute and 30 seconds after the exposure due to difficulties to get a correct probe fitting.

The ILO96 was programmed to measure DPOAEs in loop periods of either five or ten minutes. A checkfit was performed before each loop. According to the schedule depicted in figure 1, there were two loop periods of ten minutes in interval A, a loop of ten minutes in interval B and a loop of five minutes in interval C. Moreover, after the first loop of interval A, the system stopped data collection, saved the data and started a new loop. This interruption took ten seconds approximately.

#### c. Determination of the final DPOAE level

The final DPOAE level is obtained in the same way as the pre-exposure level, except that the order of *refitted* and *non-refitted* measurements was balanced across subjects depending on the order during the determination of the pre-exposure level. On average, the final DPOAE level was measured  $70 \pm 2.6$  minutes after the end of the sound exposure.

#### d. Calculation of DPOAE shift

The DPOAE shift for a particular time during the recovery is calculated as the difference between the pre-exposure DPOAE level and the corresponding post-exposure DPOAE level. Thus, a positive shift denotes a decrease in amplitude of the DPOAE. For the calculation of the DPOAE shift, data are not included if pre- and post-exposure levels are both below the noise floor. If a DPOAE level is above the noise floor before the exposure, but below the noise floor after the exposure, both values are accepted as valid.

### III. RESULTS

#### A. Hearing levels

Figure 2 shows the average hearing levels of left and right ears exposed to the 2 kHz tone. Hearing levels were measured before and 50 minutes after the end of the sound exposure. A balanced three-way-ANOVA analysis was done in order to find whether there are significant differences. The factors are: (1) determination time of the hearing level –before or 50 minutes after the sound exposure; (2) ear under measurement – left or right; and (3) frequency (6 levels). The significance level of all the

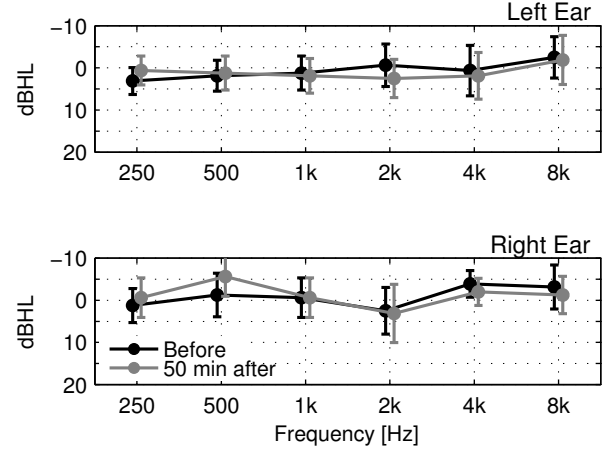


FIG. 2. Average hearing levels in the exposed ears before and 50 minutes after the sound exposure. Errorbars show the confidence interval ( $\alpha = .05$ ). Top: left ear; bottom: right ear.

statistical analysis in this paper is  $\alpha = .05$ . There are no significant differences in any of the main factors or their interactions.

#### B. DPOAE individual results

Figure 3 presents the results from six subjects and it shows DPOAE levels for one specific distortion-product frequency as a function of time during the course of the experiment. Subjects 9 and 4 show a pronounced DPOAE-shift immediately after the sound exposure. In addition, DPOAE levels from subject 9 seem to recover completely, as they become similar to pre-exposure values; whereas DPOAE levels from subject 4 seem to reach an asymptote starting at 20 minutes, approximately 2 dB below pre-exposure levels.

For the remaining subjects the following observations are derived: subjects 8 and 11 show small DPOAE shifts. Additionally, subject 8 does not show a gradual recovery. Instead, the recovery resembles a staircase. Subject 2 shows a low-level emission and there is no evident alteration in DPOAE levels after the sound exposure. Finally, subject 5 presents an unusual recovery pattern, as DPOAE levels seem to increase immediately after the sound exposure and decrease later on along the recovery – until 70 minutes after exposure, in which the fi-



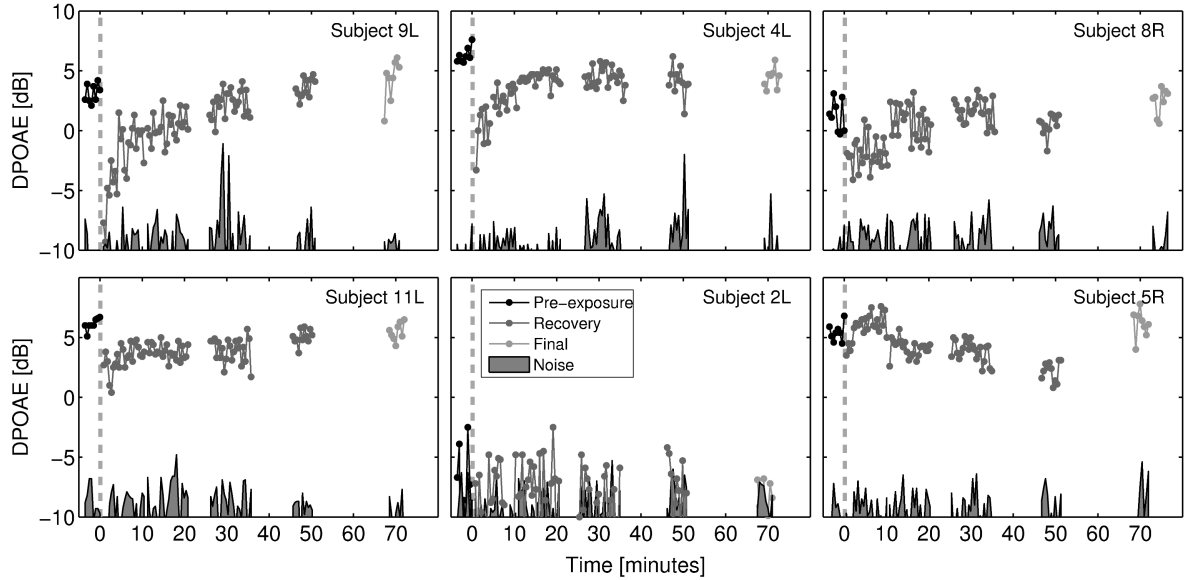


FIG. 3. DPOAE levels as a function of time for six subjects at  $2f_1 - f_2 = 1929$  Hz ( $F_{gmean} = 2739$  Hz). The ordinate shows DPOAE levels and the abscissa the time in which the DPOAE was measured. The vertical dashed-line at zero minutes marks the transition between pre and post-exposure measurements. The background noise is also shown (grey area).

nal DPOAE level appears to be greater than the pre-exposure level.

Figure 4 shows the individual DPOAE shift of the 16 subjects in the following 50 minutes after exposure. DPOAE shifts are highly individual and the impact across subjects varies both in frequency and level. In general, the biggest shift is seen within the first 20 minutes of the recovery. Some subjects show a pronounced shift at specific frequencies immediately after the exposure (e.g. subjects 7, 9 and 16), while others show a small shift (e.g. subjects 5 and 10).

### 1. Effects on fine structures

The analysis of the DPOAE fine-structures is done according to the classification algorithm proposed by Reuter and Hammershøi (2006). The algorithm monitors the ripples of the fine structures, which are characterized by a maximum DPOAE level (peak) located in between two minima (valleys). Changes in the DPOAE fine-structures are studied by plotting the maxima and minima of the ripples during the course of the experiment. A ripple is considered valid (and therefore plotted) if it satisfies two conditions: (1) the ripple height (RH), which is the level difference between the maximum and the mean of the two minima, must be higher than 3 dB; and (2) the maximum of the ripple ( $SNR_{max}$ ) must be at least 3 dB above the noise floor. These values are chosen in order to evaluate ripple characteristics with the lowest spread across subjects, and they are based on two considerations: (1) to distinguish true ripples of the fine structures from merely small variations in DPOAE levels; and (2) to avoid that ripples with minima below the noise floor might bias the analysis of the fine structures.

Figure 5 shows individual results from eight subjects. The characteristics of the fine structures are very different across subjects. Only subject 9 shows big level differences between peaks and valleys -with the greatest RH of 15 dB- and therefore the maxima and minima are easier to localize. Other subjects (e.g. 1 and 7) do not show pronounced fine structures and the peaks and valleys of the ripples cover a broader frequency range, which makes the detection of the maxima and minima not so evident. Finally, there are subjects with no measurable fine structures (e.g. subject 4).

### C. DPOAE group analysis

Figure 6 shows the average DPOAE shift across subjects as a function of time and frequency. Data from left and right ears are grouped together. Averages are calculated in intervals of 30 seconds. Intervals are accepted if they include data from at least 10 subjects. Most of the recovery occurs within the first 20 minutes after the end of the sound exposure -as the DPOAE shift drops to about 2 dB for all measured frequencies. When the frequency data in figure 6 are grouped together along the same recovery function, the DPOAE shift can be modeled with two exponential functions of coefficients  $y = 1.63 \cdot e^{-t/3.33} + 3.45 \cdot e^{-t/47.6}$  ( $R^2 = 0.77$ ).

#### 1. Pre-exposure and final DPOAE levels

Figure 7 shows the average DPOAE levels across subjects for pre-exposure and final measurements 70 minutes after the sound exposure. Next, it is studied whether (1) DPOAEs were completely recovered by the

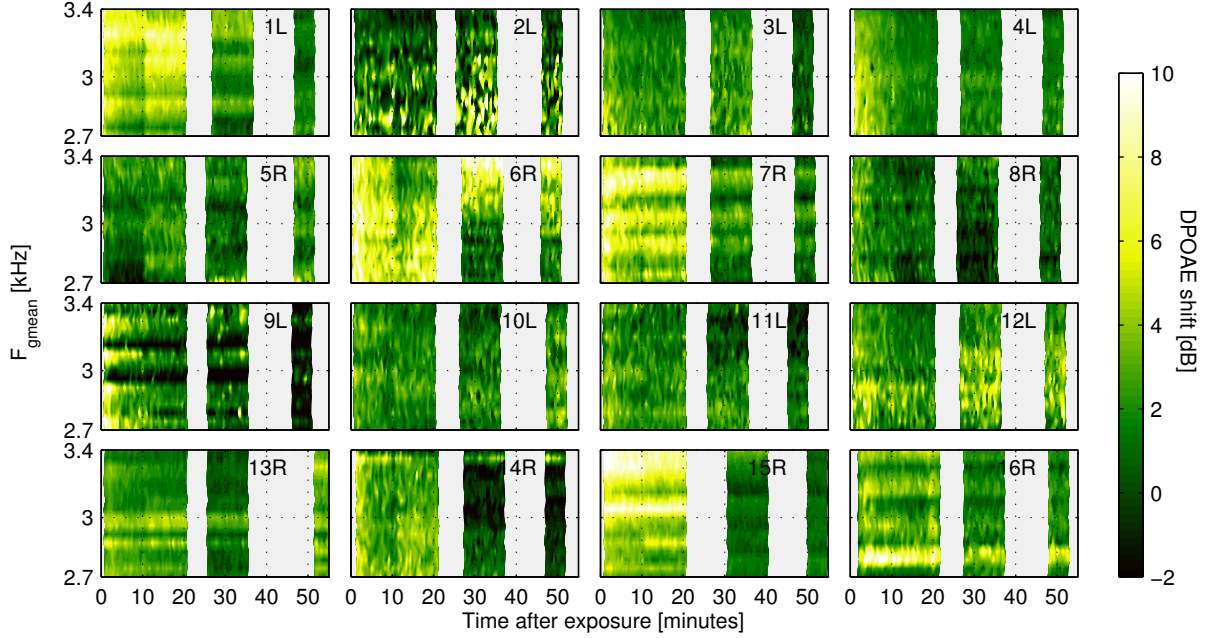


FIG. 4. Individual DPOAE shift of the 16 subjects in the following 50 minutes after the sound exposure (color online). The number and ear of the subject are given at the top of each subplot. The abscissa shows the time in minutes after the end of the sound exposure. The ordinate corresponds to the geometric mean of the primaries.

end of the experiment; and (2) reinserting the sound probe in the ear canal of the subjects influenced the determination of pre-exposure and final DPOAE levels. On average, the standard deviation of four refitted and four non-refitted measurements is similar, with a value within 0.5 – 1.5 dB – both for pre-exposure and final measurements. Statistical differences are evaluated with a 3-way ANOVA analysis of factors: (1) probe fitting –refitted/non-refitted; (2) determination time of the DPOAE level –pre-exposure/final; and (3) frequency (16 levels). The probe fitting does not result in significant statistical changes between refitted and non-refitted measurements. However, there is a weak statistical difference between pre-exposure and final DPOAE levels ( $F_{(1,975)} = 7.9$ ,  $p = .047$ ). As the p-value is almost the same as the significance level ( $.047 \approx .05$ ), it can be argued that the differences between pre-exposure and final DPOAE levels are negligible. Finally, there are no significant differences across frequencies or in any of the interactions between factors.

## 2. Stability of primary levels

Differences in the primary levels  $L_1$  and  $L_2$  might cause the pattern of the fine structures to get shifted along the frequency axis (He and Schmiedt, 1997). The stability of the primary levels during the course of the experiment is depicted in Figure 8. The figure shows the mean of the primaries, averaged across subjects and frequencies, and confidence intervals. Results are depicted before the sound exposure (*Pre*); at 10-minute intervals during the recovery –except the last interval which takes only 5 min-

utes; and at the end of the experiment (*Final*). There are no significant statistical differences in any of the intervals.

## 3. DPOAE fine structures

It is studied whether there are differences across subjects in the characteristics of the DPOAE fine structures during the course of the experiment. The following parameters are evaluated: (1) number of ripples (NR); (2) ripple height (RH); and (3) frequency specificity of the maxima and minima of the fine structures ( $F_{max}$  and  $F_{min}$ ). Figure 9 shows the mean and confidence interval of these parameters before the sound exposure; at five-minute intervals during the recovery; and at the end of the experiment. Five-minute intervals are chosen in order to have a similar number of observations between intervals. All parameters remain similar throughout the measured time span.

An unbalanced one-way ANOVA analysis of the data in figure 9 indicates that there are no significant differences for any of the parameters.

## IV. DISCUSSION

In the present study, the effect of sound overexposure on DPOAEs is highly individual. Differences across subjects are seen in (1) the magnitude of the DPOAE shift; (2) frequency specificity of the DPOAE shift; (3) the recovery pattern; and (4) the variation of the ripple height

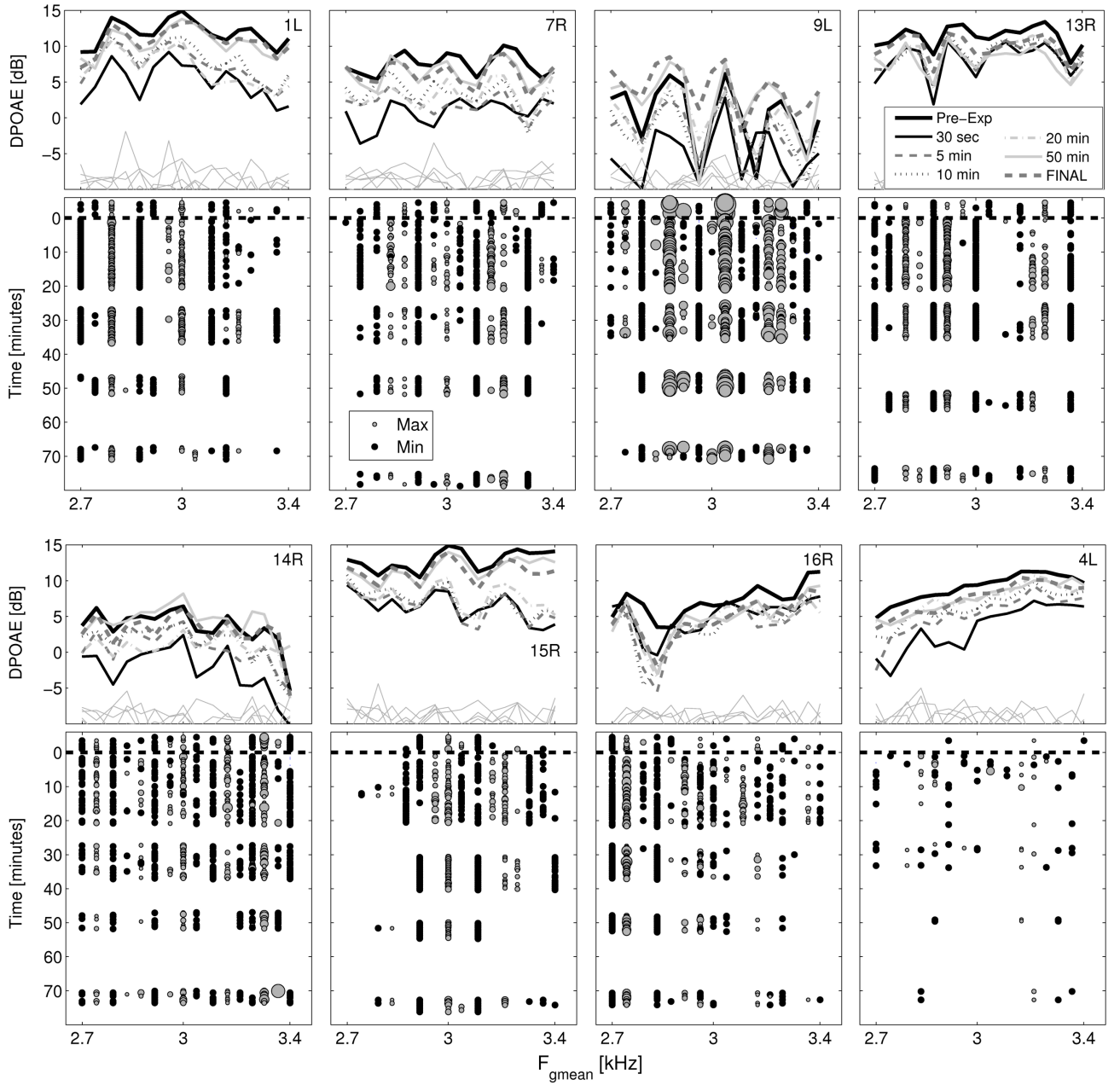


FIG. 5. DPOAE levels and effects on fine structures from eight subjects. There are two subplots per subject arranged column-wise. Top: DPOAE levels as a function of frequency at specific instants during the experiment. Bottom: evolution of the maxima and minima of the fine-structure ripples. The abscissa corresponds to the time in minutes after the sound exposure. The horizontal dashed black-line at zero minutes marks the transition between pre and post-exposure measurements. The maxima and minima of the ripples are shown as grey and black circles respectively. The size of the maxima depends on the ripple height, i.e., the bigger the circle the bigger the ripple height.

of the fine structures. This high inter-subject variability is usually seen in the overexposure effects of both DPOAE (Engdahl and Kemp, 1996; Reuter *et al.*, 2007; Marshall *et al.*, 2001) and TTS studies (Quaranta *et al.*, 1998). In our study, the individual variability can not be attributed to differences in the sound exposure between subjects because (1) calibration results show that the  $L_{ear}$  at the blocked-entrance ear canal had a standard deviation below 0.4 dB; and (2) the sound transmission

within the ear canal at 2 kHz will lead to differences below 2 dB in the SPL at the eardrum (Hammershøi and Møller, 1996). Thus, we attribute the variability to individual susceptibility to noise overexposure.

Most subjects in figure 4 show a gradual recovery of the DPOAE-shift. However, few subjects show an abrupt shift at the transition between the measurement intervals programmed in the ILO96 (e.g., subject 1 at 10 minutes). A further analysis of these subjects show that



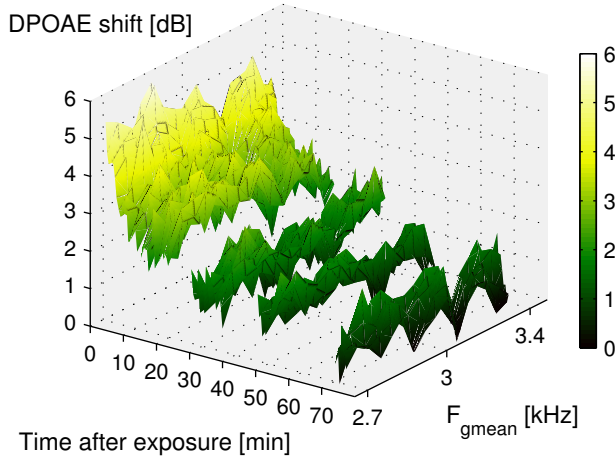


FIG. 6. Group average DPOAE-shift (color online).

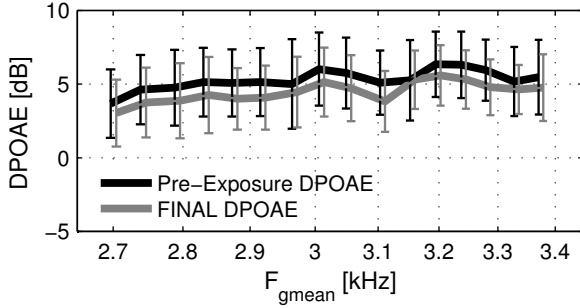


FIG. 7. Group average DPOAE levels for pre-exposure and final measurements. Errorbars show the confidence interval ( $\alpha = .05$ ).

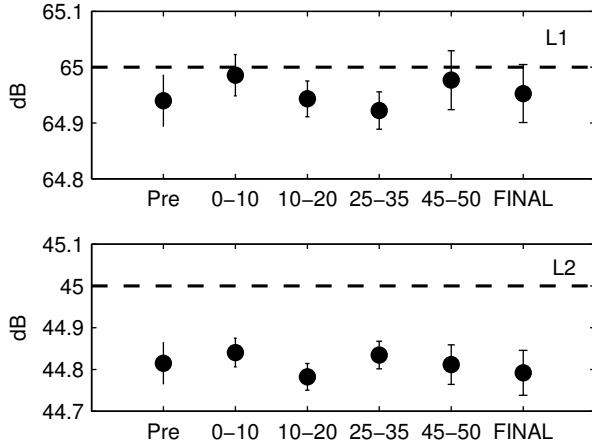


FIG. 8. Stability of the overall primary levels L1 and L2 at different intervals. The errorbars show the confidence interval ( $\alpha = .05$ ). The dashed horizontal lines show target levels of 65 and 45 dB.

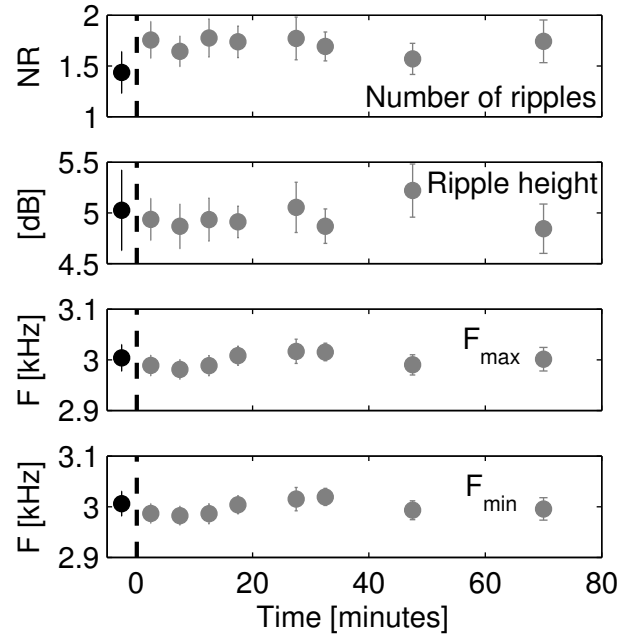


FIG. 9. Characteristics of the fine structures averaged across subjects during the course of the experiment. From top to bottom: number of ripples (NR), ripple height (RH), and frequency specificity of the maxima and minima ( $F_{max}$  and  $F_{min}$ ). Pre-exposure values are shown in black. Errorbars show the confidence interval ( $\alpha = .05$ ).

there are significant statistical differences in the primary levels  $L_1$  and  $L_2$  during the intervals, with a deviation within  $\pm 2$  dB. Therefore, the abrupt DPOAE-shift transition can be attributed to a small alteration in the probe fit during the measurement – i.e., when the ILO96 performs a checkfit prior the new macro, it re-calibrates the primary levels, compensates for the misadjustment of the sound probe and the differences are revealed in the plot. Another example of abnormal recovery is seen in subject 15. This subject shows a pronounced DPOAE shift above 3 kHz after the sound exposure and it remains constant during the first 20 minutes with no evident signs of recovery. However, the DPOAE-shift is almost non-existent after the first pause. We hypothesize whether the experimental protocol used in the experiment –i.e. DPOAE measurements are performed with high time and frequency resolution in a narrow area of the basilar membrane– might have inhibited the recovery of OHC function for this subject and thereby DPOAE levels.

The group-recovery DPOAE-shift in figure 6 shows that DPOAEs were reduced approximately 5.0 dB in the measured frequency range (2.7 – 3.4 kHz) within the first 10 minutes. This value is in agreement with the results by Engdahl and Kemp (1996), who exposed 9 subjects to a monaural narrow-band noise centered at 2 kHz with  $L_{ear} = 102$  dB during 10 minutes and measured DPOAEs in the 1 – 6 kHz range. The greatest DPOAE shift was seen in the range 3 – 5 kHz, with a maximum value of 5 dB. Due to the similarities between the sound exposure used by Engdahl and Kemp and the one used in

the present study, we consider that the frequency range studied in our experiment is the most susceptible to show the biggest impact after overexposure, and that other regions in the basilar membrane might have shown similar or lower DPOAE-shifts, if any.

This experiment shows that the fine structures are highly individual and they are not affected in a systematic manner after sound overexposure. In general, the location of the maxima and minima of the ripples remained stable during the recovery, except for few subjects who showed a downward shift in frequency at specific instants of the recovery. For subjects with smooth fine structures—for which the location of the maxima and minima is not well defined in frequency—the shift can be attributed to a quantization error of the classification algorithm (e.g., subjects 7 and 15 in figure 5). In this way, adjacent DP-points may have similar levels, and therefore, small fluctuations of the DPOAE levels may influence the either-or decision of the algorithm. This also explains why some subjects do not show well-defined maxima and/or minima for pre-exposure DPOAE levels. An exception is subject 9. This subject shows pronounced fine structures which appear randomly downward-shifted between 3.2–3.4 kHz during the recovery. Engdahl and Kemp (1996) also reported a downward shift in their two subjects. According to our results, it might be possible that their data were influenced by the determination instant in which DPOAEs were measured during the recovery. Another explanation for the downward shift in frequency is based on the influence of potential SOAEs. Furst *et al.* (1992) compared changes in SOAEs and the fine structure of hearing thresholds after intense noise stimulation. They reported (1) a temporary reduction in the SOAE frequency and amplitude; and (2) a decrease between the minimum and maximum of the threshold fine structure together with a downward shift in frequency. The authors attributed the shift to a change in the stiffness of the cochlear partition due to a reduction in the amplifier gain. That is the characteristic frequency (CF), defined as the point of maximum vibration in the basilar membrane, might depend on the action of the OHCs. A lower contribution of the OHCs due to exhaustion after noise overexposure may result in a decrease of the CF frequency. According to the results by Furst *et al.* (1992) it is possible that the frequency shift observed in the DPOAE fine structures of subject 9 might be due to the influence of a potential SOAE—in a similar manner as they reported for the hearing threshold fine structures of their subjects.

Figure 5 shows that—for some subjects—the ripple height (RH) of the fine structure can vary considerably after sound overexposure. Subjects 13, 7 and 16 show a bigger shift in the minima and an increase in the RH during the early recovery period. On the contrary, subject 9 shows a bigger shift in the maxima, resulting in a lower RH. For the rest of the subjects with measurable fine structures there are no clear differences. This variability is in agreement with the results by Reuter *et al.* (2007) and it can be explained according to the two-source model of DPOAE generation. The model considers that the fine structure is caused by constructive and

destructive interference of the distortion component  $f_2$  and the reflection component  $2f_1 - f_2$ . Variations of the RH after sound overexposure can be indicative of shifts in the relative amplitude of the two components. Our results indicate that the shift may be more acute for some subjects than others but—on average—there are no significant differences in the characteristics of the RH due to overexposure (figure 9), which suggests that the two components might be affected equally. This theory could explain why no systematic changes of fine structure could be observed in this study.

In the literature it is suggested that the individual differences in the behaviour of the fine structures can be predicted by analysing the phase of the DPOAE (Talmadge *et al.*, 1999; Long *et al.*, 2008). In this way, both the generation and reflection component can be separated and the analysis of the phase change with frequency can provide a clue as to which component is dominant, and thereby what will be the effect on the fine structures. Several methods for separation of the two components have been proposed (Shera and Zweig, 1993; Stover *et al.*, 1996; Long *et al.*, 2008). However, the methods rely on principles that still need to be validated before they can be reliably applied. One of the main constraints is that the methods assume that the cochlea is a linear and non-dispersive system. This assumption could lead to wrong estimations of the delay of the two components (Tubis *et al.*, 2000). An alternative method to separate the two components consists on adding a suppressor tone close to the distortion component  $2f_1 - f_2$  (Heitmann *et al.*, 1996; Mauermann and Kollmeier, 2004). The suppressor tone aims at avoiding the influence of the distortion component while leaving the generator component  $f_2$  unchanged. Currently, a universal suppressor level for all subjects does not exist, and this may sometimes increase the fine structure instead of reducing it (Talmadge *et al.*, 1999).

The results suggest that the fine structures of the DPOAE might not be a better indicator of early hearing loss than the DPOAE level alone. On the contrary, the variability seen across subjects might complicate the interpretation and comparison of results.

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# Is it necessary to penalize impulsive noise +5 dB due to higher risk of hearing damage?

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It is studied whether the +5 dB penalty for impulsiveness established by ISO 1999:1990 accounts for a higher risk of noise-induced hearing loss. A total of 16 normal-hearing human subjects were exposed for 10 minutes to two types of binaural industrial-recordings: (1) a continuous broad-band noise normalized to  $L_{EX,sh} = 80$  dBA; and (2) the combination of the previous stimulus with an impulsive noise normalized to  $L_{EX,sh} = 75 + 5_{dBpenalty} = 80$  dBA (peak level 117 dBC and repetition rate of 0.5 impacts per second). Distortion product otoacoustic emissions (DPOAEs) were measured in a broad frequency range before and in the following 90 minutes after the exposure. The group results show that the continuous exposure had a bigger impact on DPOAE levels, with a maximum DPOAE shift of approximately 5 dB in the frequency range 2 – 3.15 kHz during the first 10 minutes of the recovery. No evident DPOAE shift is seen for the impulsive+continuous stimulus. The results indicate that the penalty overestimated the effects on DPOAE levels and support the conception that the risk of hearing loss from low-level impulses may be predicted on an equal-energy basis.

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## I. INTRODUCTION

Industrial workers are often exposed to noise levels that can damage their hearing. The risk of noise-induced hearing loss (NIHL) can be predicted according to the International Standard ISO 1999:1990. Unfortunately, the validity of the standardized method to correctly predict the risk of NIHL for all types of noise exposures is still under question, especially for noises of impulsive character (Kardous *et al.*, 2005).

The current conception is that the risk of NIHL from all types of existing noise in industrial environments can be predicted on an energy basis as long as the peak levels do not exceed 140 dBC (Von Gierke *et al.*, 1982; Price, 1981). Therefore, ISO 1999:1990 prescribes a unified measurement method for all types of noise, also impulsive, consisting of readings of the equivalent A-weighted level  $L_{Aeq}$ , normalized to a normal working day of eight hours  $L_{EX,sh}$ . The method is based on the equal-energy hypothesis (EEH), which postulates that (1) noise exposures with the same A-weighted energy may produce similar effects on our hearing independently of their temporal and spectral properties; and (2) an increase of 3 dBA in the sound pressure level (SPL) of a noise must be offset by a halving of the exposure duration if the same equivalent level is to be maintained (Martin, 1976).

ISO 1999:1990 allows adding a +5 dB penalty to the measured  $L_{Aeq}$  if a noise is “impulsive” based on the presumption that impulsive sounds might pose a higher risk of hearing damage. The penalty is based on the results by Passchier-Vermeer (1968) showing that the hearing levels of workers exposed to widely fluctuating noises developed significantly larger losses (approximately 5 dB

higher at 4 kHz) than workers exposed to continuous levels. However, there is not yet a universally accepted approach, and the penalty for impulsiveness may vary up to 7 dB between countries depending on their noise legislation. Another uncertainty is that ISO 1999:1990 does not specify when an impulsive sound must be penalized, and the guidelines for penalization differ across countries. In Denmark –for example– the *Danish Working Environment Authority* establishes that a +5dB penalty must be added when measuring noises of impulsive character with peak levels higher than 115 dBC (or dBA) more than once per minute (Arbejdstilsynet, 2003).

The available longitudinal studies in industrial environments of impulsive character suggest that the penalty may not be necessary for all impulsive sounds. Some studies reported that the hearing loss of workers exposed to impulsive noise could be predicted according to the EEH (Atherley and Martin, 1971; Guberan *et al.*, 1971; Taylor *et al.*, 1984; Surovov *et al.*, 2001); while other studies showed that workers developed a higher loss and favored the use of the penalty (Passchier-Vermeer, 1971; Voigt *et al.*, 1980; Sulkowski and Lipowczan, 1982; Surovov *et al.*, 2001; Thiery and Meyer-Bisch, 1988). These contradictory results suggest that, up to certain limits, the EEH may be valid for all types of noise. Unfortunately, we simply do not have enough systematic data to delineate the range of conditions for which the EEH is appropriate – nor to determine when to penalize a given impulsive sound.

Most of the current knowledge regarding the vulnerability of the human auditory system to noise overexposure is based on audiometric data regarding temporary (TTS) and permanent threshold shifts (PTS). However, otoacoustic emissions (OAEs) may provide a better diagnosis tool to assess risk of NIHL (Lapsley Miller and Marshall, 2006). OAEs are a sensitive indicator of the physiological activity of the outer hair-cells (OHCs) in

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the inner ear. The function of the OHCs is essential for healthy hearing and it is believed that the onset and gradual development of NIHL is mainly a consequence of OHC loss (Saunders *et al.*, 1991). Thus, OAEs reflect vulnerability of the inner ear after acoustic overexposure due to metabolic changes in the OHCs (Kemp, 1986; Probst *et al.*, 1991). Longitudinal and cross-sectional studies have shown that OAEs can diminish before hearing levels in populations exposed to high levels of noise (an overview of the studies is given by Lapsley Miller and Marshall, 2006). For this reason OAEs may be more sensitive than pure-tone audiometry to detect incipient hearing loss. In addition, changes in OAE levels and audiometric tests have shown similarities in the frequency specificity of TTS and PTS; and in the time course of the recovery of TTS (Marshall *et al.*, 2001; Engdahl and Kemp, 1996; Emmerich *et al.*, 2000; Reuter *et al.*, 2007).

In the present study human subjects were exposed under laboratory conditions to two different noise stimuli. One of the stimuli was penalized +5 dB for impulsiveness as specified by the Danish legislation (Arbejdstilsynet, 2003). The purpose was to investigate whether there is a difference in the temporary changes from the two stimuli, which may be indicative of a higher risk of NIHL. Temporary changes in the hearing of the subjects were monitored with measurements of distortion product otoacoustic emissions (DPOAEs). The assumption is that, if the penalty is correct, both stimuli may produce similar effects on the DPOAEs. The experiment was approved by the *Danish National Ethical Committee on Biomedical Research Ethics*.

## II. MATERIALS AND METHODS

### A. Subjects

In total 16 subjects (eight males and eight females) participated in the experiment. The average age was 24.3 years with a standard deviation (SD) of 3.7 years. Subjects had hearing thresholds below 20 dBHL from 250 Hz to 8 kHz measured with octave intervals, a middle-ear pressure within  $\pm 50$  daPa, and an active stapedius reflex. None of the subjects reported known incidents of excessive noise exposures or abnormal reactions to noise, e.g. annoyance or headaches. All subjects were students at Aalborg University and they were remunerated for their participation.

### B. Noise exposures

Subjects were exposed to two different noise stimuli. Both stimuli had a duration of 10 minutes and they consisted of binaural recordings of industrial noises with the following properties:

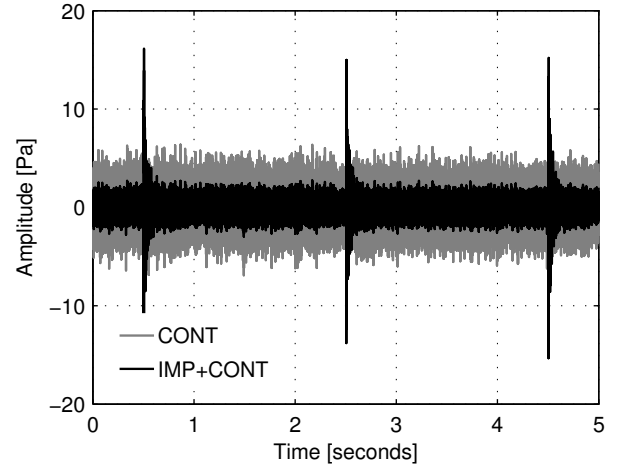


FIG. 1. Five-second excerpt of the diffuse-field waveforms of stimuli CONT and IMP+CONT.

#### 1. Continuous noise (CONT)

The stimulus CONT was industrial noise of continuous character. The stimulus consisted of the mix of a bench driller and a table saw. Both machines radiate noise of continuous broad-band noise but in different frequency ranges; i.e., low frequency for the driller and mid-high frequency for the saw. It was decided to mix the two noise sources in order to cover a broader frequency range during the exposure. Noise recordings were done separately and mixed afterwards via software. The 10 minute-duration stimulus was obtained by concatenating a segment of 30 seconds from the original recordings. A cross-fading technique was applied in order to have a smooth and unnoticeable transition between segments. The exposure level was normalized to an eight-hour working day  $L_{EX,8h}$  of 80 dBA, which corresponds to a  $L_{Aeq,10min}$  of 97 dBA (ISO 1999:1990). This level leads to a  $L_{ear,10min}$  of 102.7 dB at the blocked-entrance ear canal (ISO 11904-1:2002). The SD across subjects was  $\pm 0.7$  dB in both ears.

#### 2. Impulsive + continuous noise (IMP+CONT)

The stimulus IMP+CONT consisted of the combination of impulsive and continuous industrial noise. The continuous noise was a scaled version in level of the aforementioned stimulus CONT. The impulsive noise was a hammer beating a metal plate with a repetition rate of 0.5 impacts per second (300 impacts in total). The 10-minute-duration sequence of impulses consisted of duplicates of one single hammer-impact selected from the original recordings. In this manner, it is assured that the impulsive parameters remained constant throughout the exposure. The impact had a peak level of 117 dBC measured in diffuse field; rise time of 1.64 msec; and a B-duration of 70 msec. The exposure level was normalized to 75 dBA, which corresponds to a  $L_{Aeq,10min}$  of

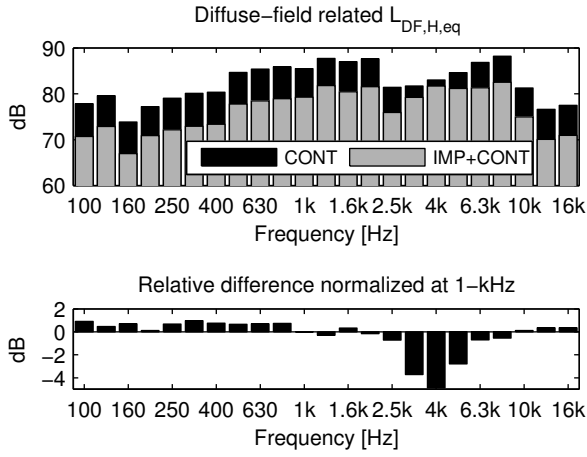


FIG. 2. Top: diffuse-field related ( $L_{DF,H,eq}$ ) in 1/3-octave bands for stimuli CONT and IMP+CONT. Bottom: relative difference between the previous levels normalized at 1 kHz.

TABLE I. Acoustic parameters of the noise stimuli CONT and IMP+CONT in a 30-second interval. All the parameters are diffuse-field related values except  $L_{ear,10min}$ , which is measured at the blocked-entrance ear canal. The crest factor is calculated as  $L_{Cpeak} - L_{Aeq,10min}$ .  $L_{AXX\%}$  are percentile levels.

param	[unit]	CONT	IMP+CONT
$L_{EX,8h}$	dBA	80	$75 + 5_{dBpenalty}$
$L_{Aeq,10min}$	dBA	97	92
$L_{Cpeak}$	dB	110	117
$L_{ear,10min}$	dBA	102.7	98.2
crest factor	dB	13	25
$L_{A10\%}$	dBA	101.5	93.5
$L_{A50\%}$	dBA	93.7	83.5
$L_{A90\%}$	dBA	79.1	60.5
kurtosis		3	32

92 dBA (ISO 1999:1990). This level leads to a  $L_{ear,10min}$  of 98.2 dB at the blocked-entrance ear canal (ISO 11904-1:2002). The SD across subjects was  $\pm 0.6$  dB in the worse ear. After penalization for impulsiveness the corrected exposure level is  $L_{EX,8h} = 75 + 5_{dBpenalty} = 80$  dBA.

### 3. Binaural recordings, sound processing and reproduction

Binaural technology is used for the recording and reproduction of real-life noise stimuli in order to reproduce realistic noise exposures in laboratory conditions. In this way, it is possible to expose subjects to the original exposure by preserving the spatial information during the recordings and reproduce the effect of proximity to the sound source. This is particularly important when considering the role of the acoustic middle-ear reflex during the noise exposures, and the fact that the incoming sound direction might have an effect on the bilateral interaction of the stapedius muscle; i.e., the contraction of the mus-

cle in one ear is influenced by the state of the muscle in the other ear (Møller, 2006).

Binaural recordings were done in a mechanical workshop at Aalborg University with the artificial head *Valdemar* (Christensen and Møller, 2000) connected to the multi-channel measuring system *Harmonie* by *01dB-Metravib*. The artificial head was placed at the position normally occupied by the operator of the machine, with the operator absent. For recordings of the hammer impacts the artificial head was placed in front of the operator so that the operator and the artificial head were symmetrically located with respect to the impact. The recordings were done with a sampling frequency of 51.2 kHz and analog-to-digital converters of 24-bit resolution. Signal post-processing was done in Matlab. Noise stimuli were bandpass filtered from 100 Hz to 16 kHz. Diffuse-field related levels ( $L_{DF,H,eq}$ ) were derived from binaural recordings according to the specifications given in the standard ISO 11904-1:2002. The original binaural recordings were down-sampled to 48 kHz for compatibility with the soundcard in the computer used for playback. Noise stimuli had a fade in of 4 seconds and a fade out of 2 seconds.

Figure 1 shows the diffuse-field related waveforms of the stimuli CONT and IMP+CONT. Figure 2 shows on top the diffuse-field-related value of both stimuli in 1/3-octave-bands; and at the bottom the relative difference between them normalized at 1 kHz. It can be seen that both stimuli have similar noise spectra except around 4 kHz due to the higher energy contribution of the hammer impact in that range. A list of acoustic parameters for both stimuli can be seen in table I. The kurtosis value suggested by Erdreich (1986) as descriptor of impulsiveness is also shown.

Noise stimuli were presented via the audiometric headphones Sennheiser HDA-200. For a correct binaural reproduction (Møller, 1992), the headphones were equalized from 100 Hz to 16 kHz according to the equalization filter described by Ordoñez (2005, appx. E).

## C. Instrumentation

### 1. Pure-tone audiometry and middle-ear test

Pure-tone audiometries were measured using the commercial audiometer *Madsen Orbiter 922* and the audiometric headphones *Telephonic TDH39*. The left ear was tested first. Hearing thresholds were determined in a frequency range from 250 Hz to 8 kHz in octave intervals by using the ascending method, which complies with the norms for automatic audiometries ISO 8253-1:1989. Tympanometries and stapedius-reflex tests were performed with the *Interacoustics impedance audiometer AT235*.

### 2. DPOAE measurements

DPOAEs were measured with the ILO96 Research-system from Otodynamics, using the operating mode

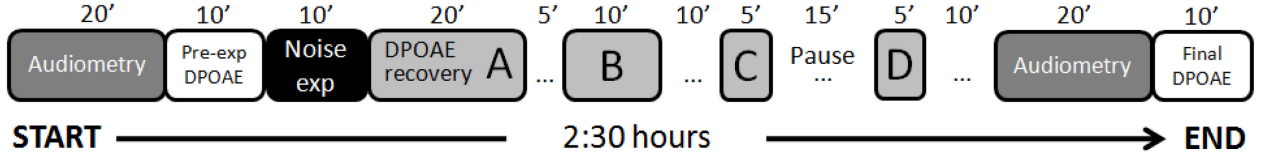


FIG. 3. Order of measurements during sessions 2 and 3. The duration of each measurement is shown in minutes.

called *DPOAE macro*. This mode is able to perform measurements in loop periods with a maximum of 16 user-defined pairs of primaries  $f_1$  and  $f_2$ . Individual macros were programmed for each subject. The only difference between macros is the presentation order of the primaries, which was counter-balanced across subjects by means of a Latin-square design.

DPOAEs were measured with a frequency ratio of  $f_2/f_1 = 1.22$  and fixed primary levels of  $L_1/L_2 = 65/45$  dB. This ratio was chosen as it may provide the largest DPOAE levels across subjects (Probst *et al.*, 1991). The choice of the primary levels is a compromise between: (1) high sensitivity to detect small changes after noise overexposure – i.e., maximum shifts in DPOAE levels can be obtained when lowering  $L_2$  compared to  $L_1$  (Whitehead *et al.*, 1995; Marshall *et al.*, 2001); and (2) measurable DPOAEs: Whitehead *et al.* (1995) measured DPOAEs for various primary-level combinations at different frequencies and reported that 65/45 dB showed relatively high level DPOAEs for all tested frequencies.

Measurements of the  $2f_1 - f_2$  acoustic distortion-product (DP) were performed with a frequency resolution of eight points per octave in the frequency range  $1282 < F_{gmean}[\text{Hz}] < 4695$  (geometric mean of the primaries). This frequency range is a compromise between avoiding low signal to noise ratio at lower frequencies, and a poor probe fitting at high frequencies. The averaging time per each pair of primaries presented was set to 1.3 seconds. This value was a compromise between minimizing random noise influence and still being able to monitor the dynamic behaviour of the inner ear after noise overexposure. With this configuration the ILO96 takes 28 seconds to measure the 16 pairs of primaries in the frequency range.

Further, a DPOAE measurement with a frequency resolution of 40 primaries per octave was performed at the beginning of the experiment. The primaries were presented ascendantly from  $905 \text{ Hz} < F_{gmean} < 5.2 \text{ kHz}$ . The purpose of this measurement was to have a detailed description of the DPOAE levels of each subject and reveal any existing fine structure of the DPOAE (Gaskill and Brown, 1990; Reuter and Hammershøi, 2006). In the following, this measurement is called *high-resolution DPOAE*.

#### D. Sessions and experimental protocol

The experiment took place at the Section of Acoustics of Aalborg University and it was divided in three sessions

–called session 1, 2 and 3. All sessions were conducted in a double-walled, sound-isolated audiometry-chamber which complies with the background noise requirements stated in ISO 8253-1:1989.

Session 1 was a screening session with the following main purposes: (1) to inform subjects about the experiment; (2) to check whether their hearing levels and middle-ear properties were normal; and to verify that (3) subjects felt comfortable during DPOAE measurements; (4) the sound probe fitted easily in their ear canal; and (5) the primary levels for the test were correctly presented. Subjects listened shortly to the sound exposure. They were allowed to decline participation after this (and at any other point in time). The high-resolution DPOAE was measured at the end of this session. The duration of session 1 was approximately one hour.

Subjects were exposed to the sound stimuli in sessions 2 and 3. Each session lasted two and a half hours. There was approximately one week in between sessions. Subjects reported that they did not go to noisy places or listen to loud music at least one day before the sessions. DPOAEs were measured only in one ear which was randomly chosen and balanced across subjects. The same ear was used in both sessions. The presentation order of the stimuli was balanced across subjects. The order and duration of the measurements is depicted in Figure 3 and it consisted of:

1. **Audiometry** ( $\approx 20$  minutes). Both ears were tested.
2. **DPOAE** ( $\approx 10$  minutes). The pre-exposure level was determined in the ear under measurement.
3. **Binaural noise-exposure** (10 minutes). Subjects were instructed to remove the headphones at any time if they felt any discomfort during the exposure. None of them did.
4. **DPOAE** (80 minutes). The recovery of DPOAE was measured in four intervals (called A, B, C and D in Figure 3) with durations of 20, 10, 5 and 5 minutes respectively. There were three pauses of 5, 10 and 15 minutes to allow subjects to relax. Pauses took place 20, 35 and 50 minutes after the exposure. Subjects stayed in the room unless they asked to go to the bathroom. Few of them did.
5. **Audiometry** ( $\approx 20$  minutes). Both ears were tested.
6. **DPOAE** ( $\approx 10$  minutes). The final DPOAE level was determined in the tested ear



## E. Measurement procedure

Subjects were comfortably seated on a chair with a head rest and they remained calm and still. Prior to each measurement the ILO96 verifies the correct fitting of the sound probe in the ear canal by a *checkfit* procedure. During the checkfit two broadband-click stimuli are alternately delivered by the two output transducers of the sound probe. The checkfit result is stored in an array and used during data collection to balance and normalize the two stimuli levels. All spectrum analyses are done by the ILO96, which performs a fast Fourier transform FFT with a frequency resolution of 12.2 Hz. The noise is estimated from the ten Fourier components nearest to but not including the  $2f_1 - f_2$  frequency. The noise is represented as all levels within two standard deviations of the background noise, i.e., the limits of the 95% confidence region.

### 1. Determination of pre-exposure DPOAE level

The DPOAE pre-exposure level is obtained as the average of eight repeated DPOAE measurements. For four consecutive measurements the sound probe was reinserted in the ear canal of the subject before each measurement (so-called *refitted*). For the remaining four measurements the sound probe was not reinserted (*non-refitted*). The presentation order of refitted and non-refitted measurements was balanced across subjects. A *checkfit* was performed every time the sound probe is reinserted.

### 2. Assessment of the DPOAE recovery

The headphones were removed immediately after the noise exposure, the sound probe was placed in the ear canal of the subject, and a checkfit was performed. On average, DPOAE measurements started  $30 \pm 8$  seconds after the end of the noise exposure. The ILO96 was programmed to perform DPOAE measurements in loop periods of either five or ten minutes. According to the schedule depicted in Figure 3, there were two loop periods of ten minutes in interval A; a loop of ten minutes in interval B; and a loop of five minutes in intervals C and D. After the first looped measurement of interval A, the system stopped data collection, saved the collected data and performed a checkfit before starting a new loop. This interruption took ten seconds approximately.

### 3. Determination of final DPOAE level

The final DPOAE level is obtained in the same way as the pre-exposure DPOAE level, except that the order of *refitted* and *non-refitted* measurements was balanced across subjects depending on the order during the determination of the pre-exposure level. On average the final DPOAE level was measured  $100 \pm 2.6$  minutes after the end of the noise exposure.

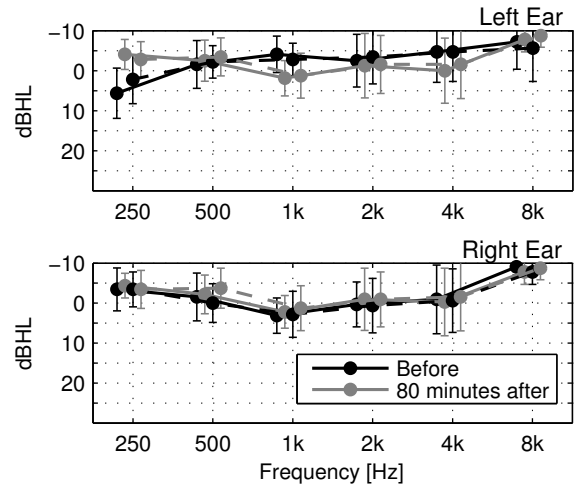


FIG. 4. Average hearing levels across subjects before and 80 minutes after the noise exposures. Continuous lines correspond to the stimulus CONT; dashed lines correspond to IMP+CONT. Errorbars show SD across subjects. Top: left ear; bottom: right ear.

### 4. Calculation of DPOAE shift

The DPOAE shift for a particular time during the recovery is calculated as the difference between the pre-exposure DPOAE level and the corresponding post-exposure DPOAE level. Thus, a positive shift denotes a decrease in amplitude of the DPOAE. For the calculation of the DPOAE shift, data are not included if they are below the noise floor both before and after the exposure. If a DPOAE level is above the noise floor before the exposure, but below the noise floor after the exposure both values are accepted as valid.

## III. RESULTS

### A. Hearing levels

Figure 4 shows the average hearing levels across subjects measured before and 80 minutes after exposure to noise stimuli CONT and IMP+CONT. Significant statistical differences are analyzed by means of a four-way ANOVA ( $\alpha = .05$  for all statistical tests in this paper). The factors are: (1) determination time of the hearing level – before/80 minutes after exposure; (2) ear under measurement – left/right; (3) noise stimulus – CONT/IMP+CONT; and (4) frequency (6 levels). There are no significant differences between hearing levels before and 80 minutes after the exposure, which means that the hearing levels of the subjects were completely recovered 80 minutes after exposure to both stimuli. The effects of the ear and type of noise are also non-significant. There is a main effect across frequency ( $F_{5,741} = 30, p < .001$ ). There is also an *ear-by-frequency* interaction ( $F_{5,741} = 6.02, p < .001$ ). This can be attributed to differences in the patterns of hearing for the left and right ears, with right ears showing a shallow U-

shaped function.

## B. DPOAE individual results

Figure 5 shows individual DPOAE results for six subjects after exposure to stimuli CONT and IMP+CONT (see figure for details). In general, results are highly individual and subjects do not show a common pattern. DPOAEs from subjects 14, 15 and 16 seem to be more affected by the *continuous* exposure; whereas subjects 1 and 13 show a slightly higher DPOAE shift for the *impulsive+continuous* exposure. In addition, subject 13 shows no evident DPOAE shift after the *continuous* exposure. It can also be seen that the most affected frequency range is different across subjects even for comparisons of the same stimulus. Subject 14 shows a significant DPOAE-shift after the stimulus CONT in the entire measured frequency range, whereas the DPOAE shift of subject 16 from the same stimulus is localized in a narrower region. Finally, subject 3 shows an abnormal recovery pattern, as DPOAE levels seem to recover during the first 20 minutes after the end of the stimulus CONT. However, there is a substantial DPOAE shift in the entire frequency range between 25–70 minutes with a value of up to 8 dB. This subject shows a similar effect at 2.1 kHz for the stimulus IMP+CONT.

## C. DPOAE group results

Figure 6 shows the average DPOAE-shift across subjects for stimuli CONT and IMP+CONT. For each stimulus the results from left and right ears are grouped together. Averages are calculated in intervals of 30 seconds. Intervals are accepted if they include data from at least 10 subjects. It can be seen that the *continuous* noise exposure has a maximum DPOAE shift of approximately 5 dB in the range  $2 < F_{gmean}$  [kHz]  $< 3.15$  during the first 10 minutes of the recovery, whereas the *impulsive+continuous* noise exposure shows no evident DPOAE-shift.

A two-way ANOVA analysis is performed to study whether there are significant differences between the DPOAE levels from stimuli CONT and IMP+CONT throughout the experiment. The factors are: (1) noise stimulus – CONT/IMP+CONT; and (2) frequency (16 levels). The analysis is done in time intervals of 5 and 10 minutes during the recovery. Differences in pre-exposure and final DPOAE levels are also evaluated. The analysis is done by comparing the DPOAE levels from both stimuli measured at the same time interval. Significant differences between the noise stimuli are observed during the first 10 minutes of the recovery ( $F_{1,10528} = 70.9$ ,  $p < .001$ ), which supports the idea that the stimulus CONT had a bigger impact on DPOAE levels than the stimulus IMP+CONT. These differences disappear throughout the rest of the recovery, except at the time interval 45–50 minutes ( $F_{1,5568} = 8.8$ ,  $p < .01$ ). There is a main effect in frequency for all the intervals ( $d.f. = 15$ ,  $F > 29.32$ ,  $p < .001$ ), meaning that subjects

have significantly higher DPOAE levels at some frequencies than others.

### 1. Sex differences

Male subjects have slightly higher pre-exposure DPOAE levels (mean $\pm$ SD,  $3.12 \pm 5.42$  dB) than female subjects ( $2.48 \pm 5.03$  dB). The results of a 2-way ANOVA analysis of factors (1) gender –male/female; and (2) frequency –16 levels, indicate that the differences are statistically significant for the two main effects and their interaction (gender:  $F_{(1,4046)} = 18.41$ ,  $p < .001$ ; frequency:  $F_{(15,4064)} = 36.91$ ,  $p < .001$ ; *gender by frequency*:  $F_{(15,4064)} = 18.56$ ,  $p < .001$ ).

During the first 10 minutes of the recovery, the stimulus CONT produced a higher DPOAE shift [dB] in female subjects ( $2.37 \pm .95$  dB) than in male subjects ( $1.58 \pm .87$  dB). According to a 2-way ANOVA analysis with the same aforementioned factors the differences are statistically significant (gender:  $F_{(1,560)} = 209.4$ ,  $p < .001$ ; frequency:  $F_{(15,560)} = 26$ ,  $p < .001$ ; *gender by frequency* :  $F_{(15,560)} = 12.3$ ,  $p < .001$ ). The exact opposite was the case for the stimulus IMP+CONT, as the DPOAE shift during the first 10 minutes was higher for male subjects ( $1.18 \pm .84$  dB) than for female subjects ( $.8 \pm .72$  dB). The differences are statistically significant (gender:  $F_{(1,560)} = 56.3$ ,  $p < .001$ ; frequency:  $F_{(15,560)} = 12.9$ ,  $p < .001$ ; and *gender by frequency* :  $F_{(15,560)} = 9.7$ ,  $p < .001$ ).

### 2. Probe-fitting effect

It is studied whether (1) reinserting the sound probe in the ear canal of the subjects influenced the assessment of DPOAE levels; and (2) final DPOAE levels were completely recovered by the end of the experimental session. The variability of DPOAE levels from repeated measurements is studied during the determination of pre-exposure levels. On average, the SD of four refitted/non-refitted measurements is below 1.5 dB for all tested frequencies. The group-average pre-exposure and final DPOAE levels from stimuli CONT and IMP+CONT are shown in Figure 7. The figure also shows the average noise level during the measurements. The difference between DPOAE level and noise level (the S/N) is between 8 and 14 dB, which suggests reliable DPOAE measurements (Lonsbury-Martin *et al.*, 1993). A balanced four-way ANOVA analysis was performed with the following factors: (1) determination time – pre-exposure/final; (2) fitting effect – refitted/non-refitted; (3) noise stimulus – CONT/IMP+CONT; and (4) frequency (16 levels). There are no significant differences for the following main effects: (1) determination time, meaning that DPOAE levels recovered completely by the end of each exposure; (2) fitting, indicating that reinsertions of the sound probe in the ear canal did not influence the assessment of DPOAE levels; and (3) stimulus. There are however significant differences across frequency ( $F_{15,1981} = 17.12$ ,  $p < .001$ ), which means that subjects have significantly

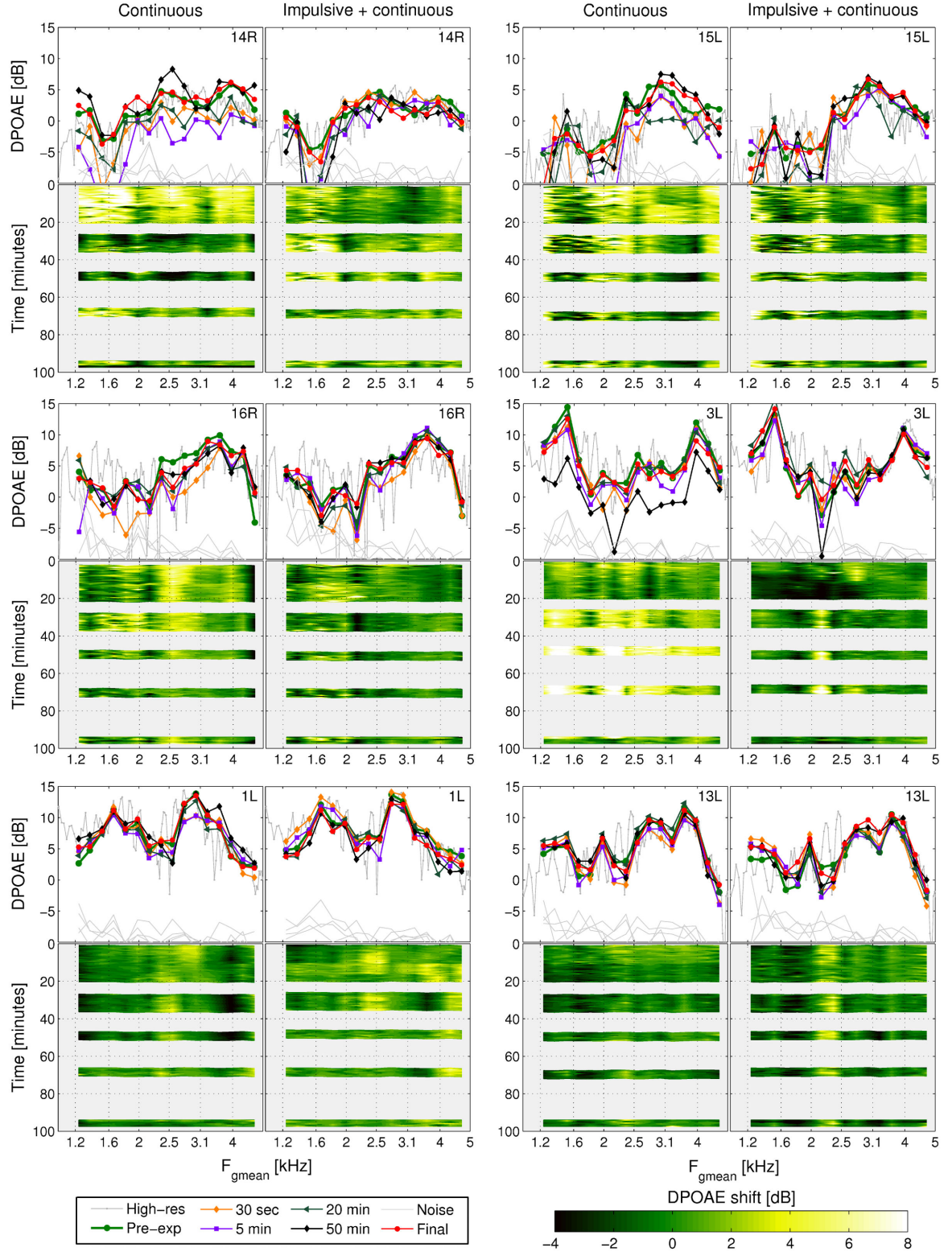


FIG. 5. DPOAE levels of six subjects after exposure to stimuli CONT and IMP+CONT (color online). The number and ear of the subject are shown at the top-right corner. Results from the stimulus CONT are presented first followed by results from IMP+CONT. The abscissa corresponds to the geometric mean of the primaries. Top: DPOAE levels at specific instants during the course of the experiment. Bottom: DPOAE shift as a function of time after the end of the exposure.



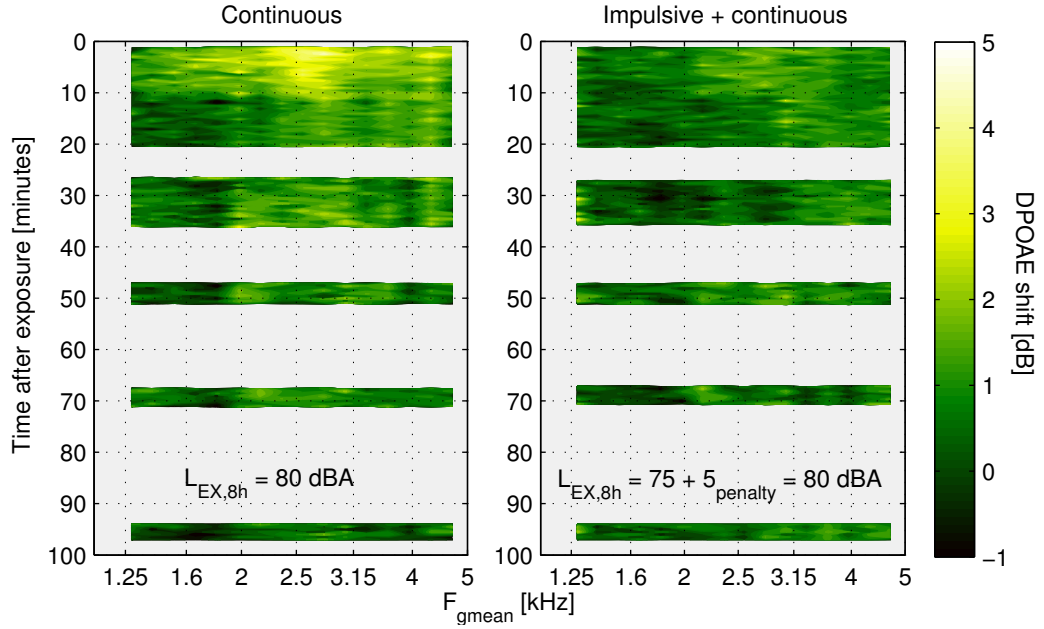


FIG. 6. Group-average DPOAE-shift from *continuous* and *impulsive+continuous* noise exposures shown as a function of the geometric mean of the primaries and the determination time after exposure (color online).

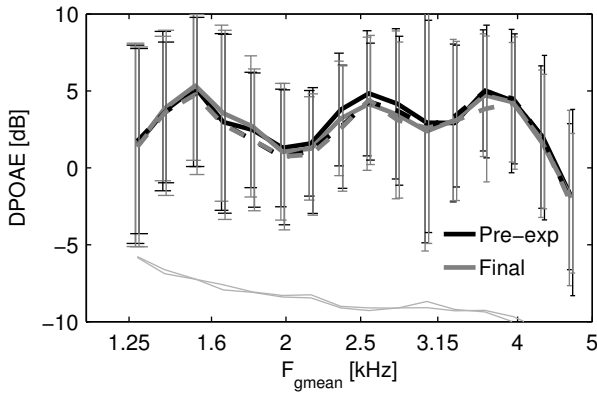


FIG. 7. Average pre-exposure and final DPOAE levels for the stimuli CONT (continuous lines) and IMP+CONT (dashed lines). Final levels were measured 100 minutes after the exposure. Errorbars show SD across subjects. The thin grey lines at the bottom correspond to the average noise levels.

higher DPOAE levels at some frequencies than others. There are no significant interactions between the factors.

### 3. DPOAE-shift comparison between maxima and minima of the fine structures

It is studied whether the maxima and minima of the DPOAE fine-structures are systematically affected in a different manner after exposure to stimuli CONT and IMP+CONT. First, it is identified for each subject whether the  $2f_1 - f_2$  DP points from measurements with

low frequency-resolution correspond to either a maximum or minimum in the fine structures of measurements with high-resolution. Next, the DPOAE-shift along the recovery of those DP points identified as maxima are selected and grouped together. The same is done for the minima. Finally, the DPOAE-shift of the maxima and minima of all subjects are combined in their respective group. In total, 61 DP-points were identified as maxima and 73 as minima. Figure 8 shows the mean DPOAE-shift of the maxima and minima from stimuli CONT and IMP+CONT. The analysis is done in five-minute intervals along the recovery in order to have similar number of observations between intervals. It can be seen that: (1) the maxima and minima from stimulus CONT show a bigger DPOAE-shift than IMP+CONT; (2) for stimulus CONT, the maxima show a slightly bigger DPOAE-shift along the recovery than the minima; and (3) for IMP+CONT there are no evident differences between the maxima and minima. Table II shows the results of a three-way ANOVA analysis from the data in Figure 8. The factors are: (1) noise stimulus – CONT/IMP+CONT; (2) DP location –maxima/minima; and (3) recovery interval –eight levels. There are interactions between the factors *noise stimulus-by-DP location* and *noise stimulus-by-recovery interval*. According to Figure 8, these interactions mean that the differences between the maxima and minima of the fine structures, as well as across intervals, are statistically significant only in the case of the *continuous* exposure. This result supports the idea that the *continuous* exposure had a bigger impact on DPOAE levels.

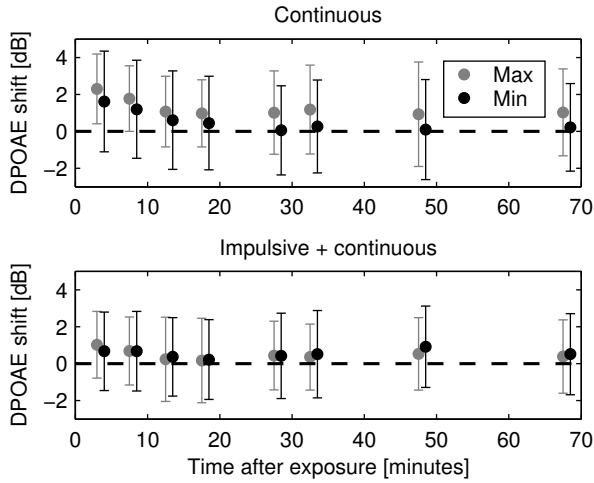


FIG. 8. DPOAE-shift comparison between the maxima and minima of the DPOAE fine structures from stimuli CONT (top) and IMP+CONT (bottom). The abscissa corresponds to the time after the end of the exposure. Errorbars show the SD across subjects.

TABLE II. Three-way-ANOVA analysis for the data in Figure 8. The factors are: (1) noise stimulus – CONT/IMP+CONT; (2) location –maxima/minima; and (3) recovery interval –eight levels.  $\alpha = 0.05$ .

factor	d.f.	F	p-value
noise stimuli	1	165.8	< .001
location	1	105.2	< .001
interval	7	55.2	< .001
stimuli * location	1	143.5	< .001
stimuli * interval	7	20.6	< .001
location * interval	7	1.8	.08

#### IV. DISCUSSION

The present study is restricted to evaluate temporary changes on the hearing of the subjects – and therefore reversible. The underlying mechanisms between temporary and permanent shifts are likely to be physiologically different, as indicated by Nordmann *et al.* (2000) for hearing thresholds and Lapsley Miller and Marshall (2006) for OAEs. In this sense, our results cannot be directly extrapolated to prolonged exposures in real industrial settings. However, the presence of a higher temporary shift in one of the stimuli may be understood as a higher risk of a permanent shift.

Individual results show that there is a high inter-subject variability on DPOAEs after exposure to stimuli CONT and IMP+CONT. These differences are seen in: (1) which stimulus produced the biggest DPOAE-shift; (2) the magnitude of the shift; and (3) the frequency specificity of the shift. As the SD in the  $L_{ear}$  between subjects is  $\pm 0.7$  dB in the worst ear, we attribute the variability in our results to individual susceptibility and not to differences in the presentation level across subjects. This inter-subject variability has also

been reported by other researchers who in similar experiments measured overexposure effects in either DPOAE (Engdahl and Kemp, 1996; Reuter *et al.*, 2007; Marshall *et al.*, 2001) or TTS (Quaranta *et al.*, 1998). It also illustrates how difficult it can be to estimate with precision the risk posed by exposure to noise on individuals, and evidences the need for statistical methods to predict the risk of NIHL in the population (Henderson *et al.*, 1993). Moreover, the statistically-significant sex differences described in section III.C.1 must be understood as a further characterization of our group of subjects. However, with our small number of subjects there is hardly sufficient statistical strength to generalize these differences to a broader population.

Four subjects show an abnormal DPOAE recovery pattern after exposure to either one of the noise stimuli or both (e.g., subject 3 in Figure 5). For these subjects the maximum shift on DPOAE levels does not occur immediately after the noise exposure. Instead, it occurs abruptly during the late part of the recovery. These abrupt increments are revealed at the beginning of each measuring macro, and they remain present during the whole measurement period of the macro. A further analysis of these subjects showed that there were statistically significant differences in the presentation level of the primaries  $L_1$  and  $L_2$  across measurement intervals, which in some cases were in the order of  $\pm 3$  dB at some frequencies. These differences indicate a misadjustment of the probe fitting during the measurements which may influence the assessment of DPOAE levels. Therefore, we attribute these abnormalities to a measurement error relating to differences in the probe fitting, and not to a physiological change in the inner ear. Further, this measurement error could be responsible for the significant difference observed between the DPOAE levels from both stimuli 45–50 minutes after the exposure in section III.C.

Group results in Figure 6 show that the *continuous* exposure (CONT), normalized to an exposure level of 80 dBA, lead to a maximum DPOAE shift of approximately 5 dB in the frequency range 2–3.15 kHz during the first 10 minutes of the recovery. Whereas the *impulsive+continuous* exposure (IMP+CONT), normalized to  $75 + 5_{dBpenalty}$  dBA, lead to no evident DPOAE shift. Therefore, the +5 dB penalty failed in predicting that the DPOAE shift from the stimuli CONT and IMP+CONT could be compared. For this reason, we conclude that for our stimuli (1) the +5 dB penalty overestimated the effects on DPOAE levels; and (2) the EEH might be a better predictor of the DPOAE shift.

Whether this conclusion can be extrapolated to other types of impulsive noise still needs to be evaluated. Nevertheless, similar results may be expected for impulses not exceeding 117 dBC peak SPL with either (1) different spectral content; or (2) presented at lower repetition rates. In the first case, impulses with a dominant spectral content in the mid frequency range (2–5 kHz), like the ones used in our experiment, are considered to be more hazardous to hearing than impulses with spectral energy concentrated at lower or higher frequencies. This statement is based on the results by Patterson *et al.* (1993), who exposed groups of chinchillas to narrow-band

impulses of different spectral content. They concluded, based on audiometric measurements and estimations of OHC loss, that impulses with spectral energy in the mid frequency range produced the highest damage. In the second case, human and animal data from both TTS and PTS studies have shown that a repetition rate of one impact per second may pose the highest auditory hazard (Trémolirès and Héту, 1980; Danielson *et al.*, 1991; Henderson *et al.*, 1991). This is because impacts within this range are not attenuated by the middle-ear reflex and pose the highest rate of acoustic stress to the inner ear. The repetition rate of 0.5 impulses per second used in this study is approximately the same as the one considered the most harmful, and therefore, lower repetition rates might not pose a higher hazard.

Results from the present study suggest that the risk of NIHL from impulsive exposures with peak levels below 117 dBC may be reasonably predicted according to the EEH. This is in agreement with the results by Surovov *et al.* (2001), who compared the hearing levels of forge hammering workers in two different industrial plants, and studied whether the degree of hearing loss could be predicted according to ISO 1999:1990. The equivalent A-weighted levels in both plants were approximately the same, but the degree of impulsiveness varied significantly. The degree of hearing loss of workers exposed to low-level impulses (113-120 dBC) could be predicted according to the standard; whereas the group exposed to higher peak levels (115-143 dBC) showed a significantly higher hearing loss. These results indicate that the peak level of the impulse may be a critical factor in the development of hearing loss. The authors concluded that the +5 dB penalty may be more suitable for noises with peak levels above 120 dBC. Our results agree with their conclusion and extend their findings to DPOAEs.

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## OPTIMIZATION OF DISTORTION PRODUCT OTOACOUSTIC EMISSION (DPOAE) MEASUREMENTS WITH THE COMMERCIAL SYSTEM ILO96

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### ABSTRACT

Measurements of distortion product otoacoustic emissions (DPOAEs) at Aalborg University are performed with the commercial system ILO96 from Otodynamics. The default measuring setup is not adequate for monitoring the recovery of DPOAEs after noise exposure because (1) data collection is interrupted repeatedly due to the operating mode of the system and (2) it is desired to have control over the presentation order of the primaries  $f_1$  and  $f_2$ , which in the default setup are always presented in an ascending sweep.

An optimized setup has been programmed with the ILO96. The setup is able to continuously measure up to 16 user-defined pairs of primaries in a loop mode. Furthermore, a systematic error was found in a particular setup of the system consisting of a mismatch between the primaries programmed to be measured and the ones actually measured. The error was reported to Otodynamics, who verified that the error occurs for this particular model.

The ILO96 is no longer under production but it is still widely used for both clinical and research purposes. Therefore, this presentation will explain the new optimized setup as well as the error in the system.

### INTRODUCTION

Measurements of distortion product otoacoustic emissions (DPOAEs) at the section of Acoustics of Aalborg University are performed with the commercial system ILO96 from Otodynamics<sup>1</sup> [1, 2, 3]. It is intended to use the system in future experiments to study how DPOAEs in human subjects are affected after over-exposure to a noise of moderate level. It is known from literature that the level of the DPOAEs is reduced after noise over-exposure and it will recover gradually depending on the severity and characteristics of the exposure [4]. Therefore, if these changes are to be monitored a fast measurement method able to continuously track the DPOAE levels in the frequency range under measurement is necessary.

The default setup of the ILO96 is adequate for measurements in which the properties of the DPOAE remain constant over the measuring period; however, it introduces some limitations when the properties of the DPOAE change with time (like as a result of noise over-exposure). Thus, the motivation behind this work has been to program a new setup with the ILO96 suitable for measurements of the recovery of DPOAE after noise over-exposure. The requirements for the new setup imply the control over the following parameters:

- Frequency value of the primaries  $f_1$  and  $f_2$  to be presented
- Presentation order of the primaries  $f_1$  and  $f_2$
- Level of the primaries  $L_1$  and  $L_2$ , and frequency ratio  $f_2/f_1$
- Possibility to collect data continuously over a specified measurement period
- The system must be able to detect fine structures of the DPOAE

Furthermore, during the optimization process a systematic error was found in a particular setup of the ILO96.

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<sup>1</sup> <http://www.oae-ilo.co.uk/enter.asp>

This manuscript starts with a description of the default setup as well as the limitations that it introduces in the assessment of the recovery of DPOAE after noise exposure. Next, the optimized setup and its features are introduced. Finally, the systematic error is explained in detail.

### DEFAULT DPOAE MEASUREMENT SETUP OF THE ILO96

The default setup of the ILO96 for measurements of DPOAE presents a pair of primaries  $f_1$  and  $f_2$  and displays the level of the distortion product located at  $2f_1 - f_2$  as it is the most prominent for most of the subjects [5]. The primaries are always swept in an ascending manner (from low to high frequencies) in a frequency range within 1-6 kHz.

In the default setup the user has control over the following main parameters:

- Level of the primaries  $L_1$  and  $L_2$
- Ratio of the primaries  $f_2/f_1$
- Averaging time per each pair of primaries presented: 0.7-10 seconds
- Frequency resolution: from one point per octave to 82 points per octave (so-called "microstructure mode")
- Distortion product to be displayed:  $2f_1 - f_2$ ,  $3f_1 - 2f_2$ ,  $4f_1 - 3f_2$ ,  $2f_2 - f_1$ ,  $3f_2 - 2f_1$  and  $4f_2 - 3f_1$

### Limitations of the default setup

The default setup introduces the following limitations when measuring recovery of DPOAE after noise exposure:

- **Operating the system requires manual navigation through different menus, which it is time consuming and interrupts data collection.** When the frequency resolution of the measurement is above 17 points per octave, the ILO96 splits the frequency range from 1 kHz to 6 kHz in smaller windows (e.g., when measuring fine structures in the microstructure mode the system needs to split the measurement in 19 windows). For each one of these windows the system will first ask the user to select the starting frequency point. Next, the system calibrates the level of the primaries to be presented and tests for the correct fitting of the sound probe in the ear canal of the subject. Then, the system starts the measurement over the corresponding window; and finally, the user needs to save the data manually. The navigation through the different windows and menus to perform the measurement results in an interruption of the data collection which is undesirable when measuring the recovery of DPOAE after noise exposure.
- **The user has no control over the presentation order of the primaries.** The level of the DPOAE data from the recovery depends on the time in which they are measured. In the default setup the primaries are always presented in an ascending sweep which means that all the DPOAE values are measured at different instants of time with the biggest time difference between the first and the last pair of primaries presented. These time differences between primaries can be eliminated across subjects if the presentation order of the primaries is counter-balanced.

### OPTIMIZED ILO96 DPOAE MEASUREMENT SETUP

The ILO96 incorporates an option called "macro mode" which allows the user to program to some extent the operations to be performed by the system. In this mode the user writes a program called "macro" by using commands defined by the ILO96. Macros are written in ASCII code with either a macro editor incorporated in the ILO96 (macedit.exe) or conventional text editors. Macros need to be saved under the file name "AUTO.MAC" which will be run automatically by the ILO96 in the macro mode.

The main features of the macro mode for the assessment of DPOAE recovery are:

- Possibility of programming up to 16 user-defined pairs of primaries
- Measurements can be done in a loop mode during a user-defined time period
- Possibility of programming  $L_1$  and  $L_2$ ,  $f_2/f_1$ , averaging time per primaries presentation (1.3-10 seconds) and the primary tones used for calibration

The main advantage of the macro mode is that the measurement process becomes almost fully-automated, and the task of the operator is more based on supervision rather than active manipulation of the system like in the default setup. However, there are two cases during the macro execution where the system awaits for the response of the operator to continue the measurement: (1) to validate the results of the calibration and probe fitting; and (2) to confirm

saving the data. In normal circumstances these two cases together will take no longer than 10 seconds and during this time data collection is interrupted.

Another advantage of the macro mode is that it introduces more flexibility in the design of a measurement protocol as it is possible to program both the values and the order of the primaries to be measured. Fine structures of the DPOAE can be measured by choosing the primaries to be presented with the appropriate frequency resolution.

The main restriction of the macro mode is that the maximum number of pairs of primaries which can be programmed is sixteen. If more primaries are desired it is necessary to save the data from the previous primaries and program a new group of (up to) 16 pairs of primaries. Furthermore, the system always calibrates the levels and checks the fitting of the sound probe before a new group of primaries is presented.

A description of the commands for programming macros can be found in the help file of the macro editor available in the ILO96. It is not the scope of this manuscript to explain all the possibilities of the macro mode; instead, we will show a simple macro example relating to the assessment of DPOAE recovery after noise over-exposure. In this example the system presents  $n$  user-defined pairs of primaries in a loop mode during a specific time "loop\_time". Then, the system saves the data in a file called "FileExample" and presents  $j$  new pairs of primaries. Both the ratio  $f_2/f_1$  and levels  $L_1$  and  $L_2$  of the primaries are also programmed in the macro.

```

. ***** MACRO EXAMPLE *****
; Everything after ";" is considered a comment by the system.
. *****
;
;
SetDataFileName(FileExample)      ; Data will be saved under the file name "FileExample"
SetFRatio(ratio)                  ; Sets the f2/f1 ratio of the measurement. The permissible range of
                                   ; the ratio is 1.05:1.8
SetExpParameters(f1,f2,L1,L2)     ; Parameters for the experiment (f1,f2,L1,L2)
                                   ; f1 and f2 determine the frequencies used during calibration
                                   ; First group of n primaries to be presented (16 max)
SetCustomFreqCount(n)
SetCustomFreq(1,f2(1))
SetCustomFreq(2,f2(2))
    :
    :                               ; Desired input value of f2 in Hz. The system will obtain the value of 1 according
    :                               ; to the ratio f2/f1 specified above
SetCustomFreq(n-1,f2(n-1))
SetCustomFreq(n,f2(n))
SetHistoryTimeOut(loop_time)      ; Total measurement time in seconds. The system will measure in a loop mode while
                                   ; measurement time < loop_time
SpectralHistory(0,0,0,0,16,2)      ; The meaning of the 6 parameters is: F1 freq shift (Hz) between each point (-120:120)
                                   ; F2 freq shift (Hz) between each point (-120:120)
                                   ; Level of F1 shift in dB for each point (-5:5)
                                   ; Level of F2 shift in dB for each point (-5:5)
                                   ; Number of subaverages (16 = 1.3 seconds, 128 = 10 seconds)
                                   ; If "2" the system will use the custom frequencies defined above
DataFileSave                      ; Saves data as "FileExample01"
;
SetExpParameters(f1',f2',L1',L2') ; New set of parameters for the following group of primaries
;
SetCustomFreqCount(j)              ; Second group of j primaries to be presented (16 max)
SetCustomFreq(1,f2'(1))
SetCustomFreq(2,f2'(2))
    :
    :
SetCustomFreq(j-1,f2'(j-1))
SetCustomFreq(j,f2'(j))
SetHistoryTimeOut(loop_time2)      ; Total measurement time in seconds. The system will measure in a loop mode while
                                   ; measurement time < loop_time2
SpectralHistory(0,0,0,0,16,2)
;
DataFileSave                      ; Saves data as "FileExample02"
;
EndMacro

```

The minimum averaging time per primaries presentation is 1.3 seconds. With this averaging time the ILO96 takes approximately 28 seconds to measure 16 pairs of primaries.

## SYSTEMATIC ERROR IN THE “SPECTRAL HISTORY MODE” OF THE ILO96

A systematic error was found in a particular setup of the ILO96. The error consists of a mismatch between the primaries programmed to be measured and the ones actually measured when using the so-called “Spectral History Mode”. This mode offers the operator more control and flexibility than the default setup and it is more suitable for DPOAE experiments in which is desired to perform cyclical measurements changing specific parameters in each cycle. It was decided to study the possibilities of this mode for the optimization of the assessment of DPOAE recovery, especially because it introduces the advantage that the system does not need to split the measurement in smaller windows; which saves considerable time.

Under this mode, the operator introduces the values of  $L_1$ ,  $L_2$  and ratio  $f_2/f_1$ . Moreover, the operator needs to specify two starting primaries  $f_1$  and  $f_2$  and the increment in Hz for consecutive primaries (in a range from  $\pm 12$  to  $\pm 120$ Hz). The error occurs when the operator enables the option “constant ratio  $f_2/f_1$ ”; which means that during the presentation of the primaries the system will increase the value of  $f_1$  depending on the increment value selected before and will obtain the new value of  $f_2$  according to the ratio  $f_2/f_1$ . The error occurs because the ILO96 does not update the value of  $f_2$  during the presentation of the primaries; however, in the saved data it appears that it did.

The error is illustrated in figure 1 by showing different steps in the measuring process ordered from one to four. First, the system presents the first pair of primaries  $f_1'$  and  $f_2'$ , measures the distortion product corresponding to  $2f_1'-f_2'$  and saves the data. Next (subfigure 1.2), the system is expected to increment the primaries to a new pair of primaries  $f_1''$  and  $f_2''$ . However,  $f_2''$  is not incremented even though the system saves that the values of the primaries were incremented correctly (subfigure 1.3). Instead, the system uses the previous value of  $f_2'$  to perform the measurement at  $2f_1''-f_2'$ . Note that the error results in a measurement with a smaller frequency ratio even though the saved data indicates that the primaries used were the correct ones (subfigure 1.4). The error is carried all along the measurement.

This error can be critical especially for experiments studying the fine structures of the DPOAE because changes in either level or ratio of the primaries can make the fine structures appear shifted [6].

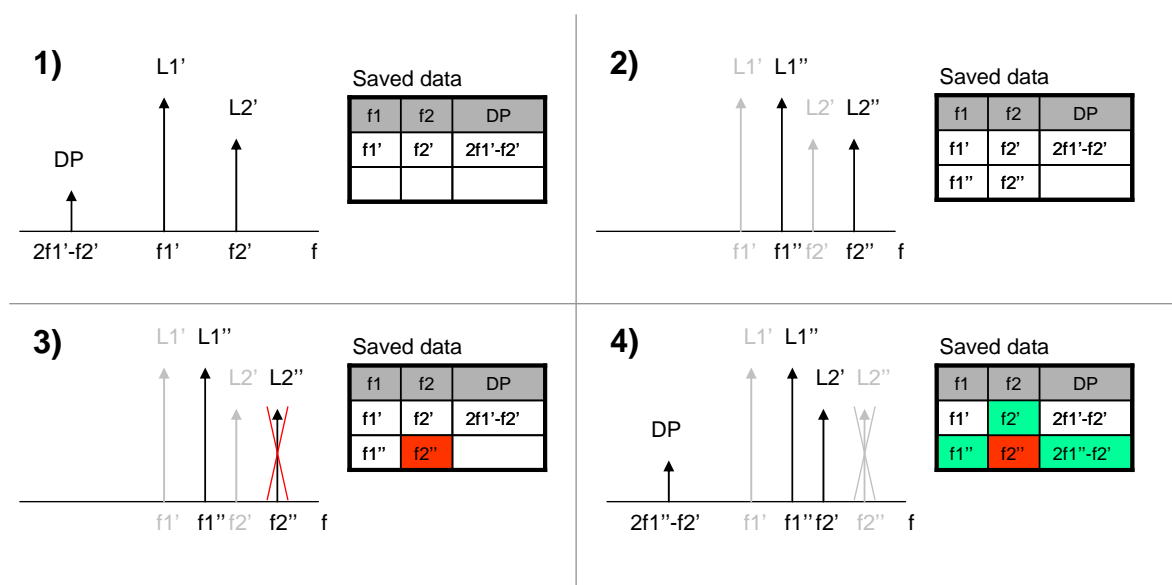


Figure 1.-Primaries presentation error in the “Spectral History Mode” when the option “constant ratio  $f_2/f_1$ ” is enabled

## CONCLUSIONS

The macro mode of the ILO96 is more appropriate for the assessment of DPOAE recovery after noise over-exposure rather than the default setup. The reason is because the macro mode allows almost full automation; it offers more flexibility in the design of a measuring protocol and it is able to measure DPOAE data continuously.

The main features of the macro are:

- Possibility of programming up to 16 user-defined pairs of primaries
- Measurements can be done in a loop mode
- Possibility of programming  $L_1$  and  $L_2$ ,  $f_2/f_1$ , and averaging time per primaries presentation
- With the minimum averaging time of 1.3 sec the system takes approximately 28 seconds to measure 16 pairs of primaries
- Possibility to measure fine structures of DPOAE by choosing the primaries with the appropriate frequency resolution

On the other hand, there is a systematic error in the "Spectral History Mode" of the ILO96 when the option "constant ratio  $f_2/f_1$ " is enabled. The error consists of a mismatch between the primaries programmed to be measured and the ones actually measured. More precisely, the value of the primary  $f_2$  for a measurement  $n$  should be the value of the measurement  $n-1$ . The error has been confirmed by Otodynamics.

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